The Journal

OF THE
AMERICAN ASSOCIATION
OF NURSE ANESTHETISTS

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The Journal of the American Association of Nurse Anesthetists

VOLUME XVI MAY, 1948 NUMBER ONE

An Open Letter to the Membership

Dear Nurse Anesthetist:

Are you an excellent anesthetist who gets nowhere? An Unknown Lady in White? If you are, it is time to take inventory of your rating in personal relations.

Unfortunately, for any of us in the professional field, our only stock-in-hand is ourselves—and we are very poor advertising agents. In no other field does one find so little effort expended in the sale of so valuable a commodity. Even the poorest merchant sees to it that his wares are put before the public, and he makes an earnest attempt to do so in a manner at least as good as his fellow merchants'.

You, Miss Anesthetist, are your "stock-in-trade." It is within your power to sell yourself into a better and fuller life. The best advertising agent in the world is Personal Relations, Unlimited. The cost is nominal—a little extra effort. The return dividend is rewarding.

Let us assume that you are willing to pay the price and sincerely want the dividend. You are now ready for that personal inventory.

You are an excellent anesthetist. Do you give your patient the opportunity to know that it is you who have given that vital service? Most patients dread the anesthesia more than the operation. Anything you can do to relieve that dread has a value beyond price. Are you one of those very efficient persons who have no friendly, personal word to say to the patient before the operation? Do you make a postanesthesia visit to show that your interest in him is real, that his recovery is of concern to you? Does he have the opportunity, after his system is clear of drugs, to meet you so that to him you may be a real person with a definite personality? If you fail here, you are passing up one of your most valuable mediums of advertising.

You have been working with your surgeons for some time. Are you still just the anesthetist—any anesthetist? Or does each surgeon know you by name? Are you a personality, or just someone performing a necessary function? If you are a personality, are you a pleasant and co-operative one? If you aren't known to your surgeons, tomorrow is not too early to start working at being a person. Fortunately, all surgeons are human, and a pleasant and co-operative anesthetist with ability always has the advantage over one who is merely efficient.

You must work closely with the operating room staff. Each nurse

in the operating room has more than her share to do. Do you see to it that you carefully refrain from adding to her load? Do you show appreciation by being agreeable and co-operative, or are you arrogant and thoughtless? Unless you are helping her by being at least co-operative, you are losing a valuable ally. She, who sees you and your conduct of the anesthesia, can raise your rating with other hospital personnel. By her manner she can assure your patient that you are the best anesthetist in the world.

Part of the success of the anesthesias you conduct rests with the nursing personnel. When you take the patient to the ward, are you dictatorial or unreasonable in your demands? Are the nurses eager to assist you, or do they dash into a room to avoid being with you for even a short time? If so, you are again failing to use a valuable means of promoting yourself. Those nurses are with the patient a great deal, and they can do much to make him appreciate your services. Praise or acknowledgement of you as a person and an anesthetist will encourage the patient to consider more closely the value of the service that you have rendered.

In the auxiliary services, the aides, corpsmen, maids, porters, and elevatormen all come in contact with you. Do you treat them with consideration and courtesy, or are you impatient and rude because they are not always aware of your needs? They respond quickly to a kind word or act. Though they cannot, perhaps, appreciate you as an anesthetist, they can help make you known as a person.

The function of the hospital, of which you are a part, is determined by the administrative personnel. Do you obey rules and regulations, or do you think of yourself as being above them? Do you attempt to understand the problems of other departments? Do you co-operate in institutional endeavors, or do you not consider them your job? Unless you are thinking and acting as though you and the hospital were making a unified effort to render service, you are failing not only the institution but yourself.

As a person you are a member of the community? Do you belong to organizations other than professional? Do you make an effort to belong to clubs or groups to which you can make a contribution as a person? Do you show an interest in community projects? Unless you do these things, you are failing society and yourself. Each person has some talent other than that he applies in his vocation. By means of your vocation you eat, but by means of an avocation you also live.

Now that you have made this personal inventory and have discovered your assets and liabilities, invest, and watch your dividends come in.—VERNA E. BEAN, R.N., President, New York State Association of Nurse Anesthetists

ANESTHESIA FOR THE SURGICAL TREATMENT OF CYANOTIC CONGENITAL HEART DISEASE

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The surgical treatment of patients with tetralogy of Fallot or similar disturbances of circulatory physiology has been a challenge to the anesthetist. Harmel and Lamont [1] reported their findings during anesthesia administration in the first 100 cases at The Johns Hopkins Hospital. The present communication reports our experience in the 475 succeeding cases and describes our methods and practices. The anatomy and physiology of this condition have been fully discussed in the literature.[2-8] A brief discussion is included here by way of review.

Most of these cases fall into the most commonly observed cyanotic type of congential heart disease known as tetralogy of Fallot. The components of this complex are (1) pulmonary stenosis or atresia, (2) interventricular septal defect, (3) dextroposition or overriding of the aorta, (4) right ventricular hypertrophy.

The symptoms and signs associated with the disease are direct consequences of the disturbance of normal circulatory dynamics. Cyanosis, the outstanding sign, results from the constant admixture of unoxygenated blood from the right ventricle with systemic blood. This admixture occurs because the stenosis produces an increase in right ventricular pressure, causing a shunt of venous blood through the interventricular septal defect and into the overriding aorta. The stenosis also reduces the pulmonary blood flow.

Cyanosis may be present at birth, or it may not develop for several months or years. At first the cyanosis may be apparent only on exercise. Later in life it is usually constant. It is often

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2. Blalock, Alfred, and Taussig, Helen B.: Surgical treatment of malformations of the heart. J.A.M.A. 128: 189-202, May 19, 1945.
3. Blalock, Alfred: Physiopathology and surgical treatment of congenital cardiovascular defects. Bull. New York Acad. Med. **22:** 64-79, 1946.
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6. Taussig, Helen B., and Blalock, Alfred: Observations on the volume of the pulmonary circulation and its importance in the production of cyanosis and polycythemia. Am. Heart J. **33:** 413-419, April, 1947
7. Blalock, Alfred: Technique of creation of an artificial ductus arteriosus in the treatment of pulmonic stenosis. J. Thoracic Surg. **16:** 244-254, June, 1947.
8. Bing, R. J.; Vandam, L. D., and Gray, F. D., Jr.: Results of preoperative studies in patients with tetralogy of Fallot. Bull. Johns Hopkins Hosp. **80:** 121-141, Feb., 1947.

associated with periods of unconsciousness. A number of patients have suffered cerebral accidents as a result of the polycythemia. Many of these patients give a history of typical "attacks." The attacks may be manifested by extreme weakness, dyspnea, loss of consciousness, or convulsions. (Morphine given intravenously has been found to be a specific treatment for an attack.) The majority of "blue babies" have a marked incapacity for any but mild exercise. Some young patients have never learned to walk; others can walk only fifty feet before requiring a rest period. Almost all patients of the group assume a typical squatting position when resting. The explanation for this has not been found.

The patient is usually small and undernourished for his age, with cyanosis of the lips, ears, fingers, and toes. Clubbing of the digits is characteristic. The pulse is slow, and the blood pressure is low with a narrow pulse pressure. Frequently blood pressure determinations may be made only by palpation. The mucous membranes are a deep mulberry color, and the conjunctivae are suffused. Polycythemia is of usual occurrence, with erythrocyte counts from normal to 12,000,000 and hematocrit readings as high as 80. Oxygen saturation of the arterial blood varies from 12 to 90 per cent.

The purpose of the operation is to create an artificial ductus arteriosus between the systemic and the pulmonary circulations, by-passing the point of stenosis. This permits some of the incompletely oxygenated blood of the systemic circulation to be recirculated through the lungs. An anastomosis is constructed between the proximal end of the divided subclavian artery or some other branch of the aortic arch, or the arch of the aorta itself, and the side of the right or the left pulmonary artery. This allows systemic blood through the ductus to reach both lungs and is therefore the preferred type of anastomosis. The subclavian artery arising from the innominate artery is considered the ideal vessel, but often one must use the carotid, the innominate, or the subclavian artery arising from the aorta. Moreover, in some instances, it is imperative that the systemic vessel be joined in end-to-end fashion to the distal end of the divided pulmonary artery. The end-to-end type of anastomosis permits blood flowing through the ductus to reach one lung only. During construction of the anastomosis, while the pulmonary artery is occluded, the patient may become deeply cyanosed. Color immediately improves with release of the occluding clamps and may be almost normal at the close of operation.

Premedication

Considerable tact, patience, and understanding are called for in caring for these patients, most of whom have been pampered and spoiled at home. A preliminary visit by the anesthetist is desirable. Thus the patient recognizes a friend when he comes to the operating room. The inability of these patients to meet an increased demand for oxygen necessitates careful consideration in

the selection of premedication. Fright, crying, or any slight exertion may produce dyspnea, increased cyanosis, and a typical attack of the type previously described. Premedication is individualized and planned so that the patient will be brought to the operating room in a drowsy state with a minimum of respiratory depression. Morphine and atropine, administered one hour and a half before the start of anesthesia, continue to be the drugs of choice. The dose of morphine is computed on a basis of 1 mg. per 5 kg. body weight, plus 1 mg. additional for the older patients.[9] Atropine is administered in an approximate ratio of 1/20 of the morphine dosage, up to 0.6 mg. Hyperpyrexia from heat retention has been a complicating factor in a non-air-conditioned operating room during hot weather. Under these conditions, atropine is administered in minimal doses.

Nembutal has not been administered as premedication to any patient in this series. A few patients have received scopolamine in place of atropine, but no marked advantage has been noted, and atropine continues to be employed routinely. Conceivably scopolamine would be equally satisfactory.

ADMINISTRATION OF THE ANESTHETIC

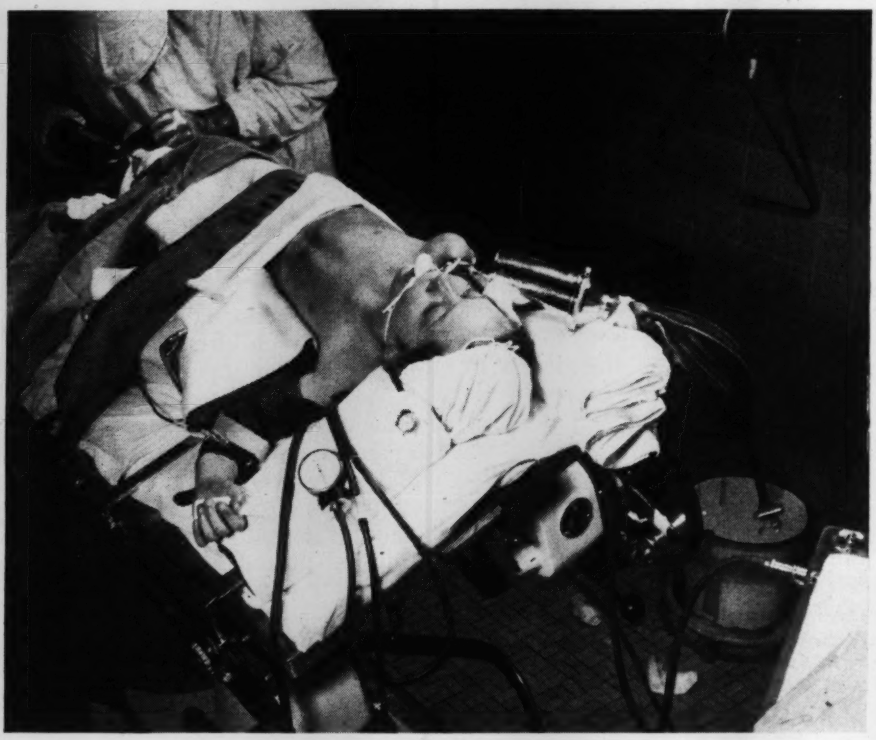
The Waters model Foregger gas machine has been used in every case. Induction has been by the semi-closed method. The to-and-fro absorption, intratracheal technic has been employed for maintenance. Cyclopropane-oxygen or cyclopropane-ether-oxygen are the preferred agents. Those patients who have heart block or some degree of cardiac failure have been anesthetized with cyclopropane-ether-oxygen until intubation has been accomplished. Thereafter anesthesia has been maintained with an ether-oxygen mixture.

Lank[10] reported that infants and small children have a high tolerance for cyclopropane. We have found this to be particularly true of those in the group with cyanotic congenital heart disease. A high percentage mixture of cyclopropane-oxygen, 50-60 per cent, is employed during the induction period. Not infrequently a higher percentage of cyclopropane is administered for a brief period. As soon as the patient has lost consciousness, the cyclopropane flow is decreased to make a 30-35 per cent mixture with oxygen. If there is marked slowing of the pulse or if arrhythmia develops, the cyclopropane flow is further reduced, and ether is added to the mixture.

Intratracheal intubation, by the direct method, is carried out as soon as sufficient relaxation of the jaw has been obtained, usually in from five to ten minutes. The catheter is secured in place by means of a tape run under the neck and tied over the mouth. A bite block of tightly rolled gauze is inserted between the teeth. The catheter is connected to a canister adapter by a curved Rovenstine catheter slip joint. As short a connecting link as possible between catheter and canister is desired in

9. Harmel, M. H.: Unpublished data.

10. Lank, Betty E.: Cyclopropane anesthesia in infant surgery. J. Am. Assn. Nurse Anesthetists 15: 3-11, Feb., 1947.



Showing anesthesia connections and position of patient for surgery.

order to reduce respiratory resistance and dead space to a minimum. The anesthetic delivery tube from the machine is slipped over the outlet nipple on the canister adapter. A spring-type paperclip closes the tail of the rebreathing bag. This affords a ready means of spilling anesthetic gases from the bag.

The intratracheal catheters used are McKesson, Magill type, or Portex plastic tubes. We found it difficult to obtain satisfactory tubes for infants in the earlier cases. We have since been able to procure the plastic tubes in sizes from 00 to 9, which have been most satisfactory. A catheter is selected of a size that fits the glottic opening snugly. Our practice is to pick out a tube which we believe will be satisfactory, plus one tube a size smaller and one a size larger. No packs are used, and rarely is a cuffed tube employed, except for adults.

After intubation and stabilization of the anesthesia, the patient is placed in position. Surgical approach is usually from the side opposite that on which the aorta descends. The patient is brought to the side of the table and the chest elevated slightly with one or more folded towels. The table

is tilted 15 to 20 degrees in the direction opposite the side of surgical approach (see figure).

Through a small incision a cannula is placed in an ankle vein on all patients after the anesthesia has been established and before the incision is made. The cannula is kept patent by a slow infusion of 5 per cent glucose solution, which is replaced by plasma shortly after the start of the operation. Whole blood replaces the plasma if the patient is anemic or if there has been excessive loss of blood during the operation. Replacement therapy is used only to the extent necessary to maintain an efficient blood pressure. Caution is taken not to overload the vascular bed, which would place additional strain on an already severely taxed heart.

Lower or mid first plane anesthesia has proved to be adequate except in rare instances. With the establishment of the desired plane of anesthesia, the cyclopropane is adjusted to a rate of flow approximating 70-100 cc. per minute; the oxygen flow rate approximates 300-400 cc. per minute. Ether is admixed as required. Small amounts of ether have been administered with the cyclopropane in an effort to provide protection from cardiac arrhythmias during anesthesia.

We have preferred to "assist" respirations by gentle rhythmic compression of the breathing bag on inspiration rather than to employ the so-called "controlled respiration" technic. The "assist" technic provides minimal movement of the mediastinum. Occasionally, mediastinal movements cannot be reduced, either by increasing the depth of anesthesia or by resorting to the "controlled respiration" technic. If this is true, a small dose of morphine is administered intravenously with good results. Seldom has this been necessary in this series.

A water manometer is incorporated in the anesthesia circuit at the machine end of the delivery tube. It may be set for any pressure desired. The fit of the intratracheal tube will influence pressure settings. Although pressures are usually set at 4-6 cm. of water for infants or small children and 6-12 cm. of water for older children or adults, we rely on a sense of touch and visual observation of the lung to determine the pressure settings.

When the pleural cavity is entered, the lung is allowed to collapse gradually to that point which will permit exposure of the pulmonary vessel. Partial inflation of the lung is sustained throughout the operation. Before the pulmonary artery is occluded for the performance of the anastomosis, the lung is fully expanded and the patient given a rest period of one to three minutes. The lung is then partially collapsed and the anastomosis started. Upon completion of the anastomosis the occluding clamps are removed, the mediastinal pleura is sutured, and the lung re-expanded and maintained in expansion until the operation has been completed. Respiratory "assist" is discontinued with chest closure. Thereafter the patient is allowed to breathe against a low positive pressure with occasional assistance of

respirations to insure adequate alveolar ventilation.

The breathing bag is emptied of any ether mixture when the anastomosis is opened. The bag is then refilled with oxygen and a low percentage mixture of cyclopropane. Helium is added to the gases within the bag after the intercostal sutures have been placed. The lungs are kept in expansion during wound closure by adjusting the flow rate of gases to the pressure set on the manometer. The helium is replaced by compressed air at the start of skin closure, and the oxygen tension is reduced in order to approximate atmospheric conditions within the alveoli. Management of the anesthesia is regulated to insure active reflexes at the completion of the operation. Many patients are sufficiently conscious to answer questions or respond to simple directions before leaving the operating room. It is reassuring to have the patient move all extremities immediately after operation.

If, for anatomic reasons, the innominate or carotid artery must be used for the anastomosis, the table is placed in a 10 or 15 degree Trendelenburg position before the vessel is occluded, in an effort to assist the blood flow to the brain.

Each patient presents a different problem. The anesthesia must be individualized in each case. Percentage mixtures, flow rates, and pressure settings are mentioned only as over-all averages. There can be no hard and fast rules to govern the management of anesthesia for this type of surgery. It has been observed that many patients whose exercise tolerance is far greater than one might expect from the degree of arterial oxygen saturation are the ones who frequently present difficulties during anesthesia.

The blood pressure is usually low and the pulse pressure narrow in these patients. Frequently, the blood pressure may not be obtainable by auscultation preoperatively. The high viscosity of the blood may account for this. The sounds may become audible during anesthesia, but in the majority of these cases determinations must be made by palpation throughout the operation. Pressures tend to fall slightly during the performance of the anastomosis. As a rule, the systolic pressure rises gradually and the pulse pressure increases after the occluding clamps have been removed. There has been no consistent blood pressure pattern in this physiologically bizarre group of patients.

Complications

Bradycardia, of varying degree, is probably the most frequently encountered complication. This may occur at any time during the anesthesia for a variety of reasons. Anesthesia is undoubtedly a prime contributing factor. When this is believed to be the case, the flow of cyclopropane is reduced immediately, or the anesthetic mixture is completely emptied from the bag. Improvement in cardiac action is usually prompt. A small number of patients react poorly to cyclopropane. These are maintained on an ether-oxygen mixture. Whenever possible, we prefer to use cyclopropane-oxygen anesthesia with only small

amounts of ether added as a prophylactic measure. It has been found easier, in our hands, to provide minimal mediastinal movements, in first plane anesthesia, with cyclopropane-oxygen rather than with ether-oxygen alone.

The heart frequently slows when the pleural cavity is entered. Atropine in small doses (0.025-0.3 mg., with an average of 0.1 mg.) is administered intravenously if the bradycardia is marked or appears to be persistent. We have found atropine a satisfactory vagal blocking agent. Only the minimal dose required to produce the desired result is employed. It may be repeated if necessary. A larger dose administered at this time results in considerable tachycardia.

Exploration or traction on the pulmonary artery may cause a slowing of the heart. A short pulmonary artery which divides early increases the degree of traction necessary and presents a surgical problem. An increased oxygen tension affords some benefit. If atropine has not been previously employed, it may be administered to advantage at this time. The exposed vagus nerve has been injected with 2 per cent procaine, 1 cc., in a few instances of cardiac hyperirritability with indeterminate results.

Cardiac arrest may occur suddenly or with only slight warning. We have been fortunate in having direct tracing electrocardiographic records made on more than 300 patients during operation. A study of these graphs indicates that there may be a definite pattern warning of impending danger.[11] Results of these studies will soon be published.

Slowing of the heart to the point of complete arrest was encountered many times in the early cases. A small group of patients had cardiac arrest from once to as many as five times during operation. At the first hint of cardiac arrest, the breathing bag is completely emptied of the anesthetic mixture and refilled with oxygen. Slightly more forceful assistance of respirations is instituted. Gentle massage of the heart muscle may be sufficient to restore cardiac action, but more intensive measures may be required. In the latter case, adrenalin, 0.25-1 cc., is injected intracardially, as well as coramine, 1 cc., and more vigorous massage of the heart is continued. Medication is repeated as often as deemed necessary. Lanatosid C (Cedilanid) may or may not be administered intravenously after the heart action has been restored. This is determined by the attending cardiologist. Our experience with procaine given intravenously has been very limited. It has been administered too late to offer conclusive evidence as to its value for these patients.

Close visual observation of the heart itself is the best means of detecting signs of impending cardiac arrest. A sudden fall in blood pressure, however, is almost always a dependable warning sign. If a change in cardiac tonus or action is noted promptly, the immediate administration of atropine intravenously may obviate a more serious crisis. Cerebral damage is almost certain to ensue if cardiac action is not restored quickly. If the heart

11. Ziegler, R. B.: Personal communication.

action ceases more than twice during operation, it is probable that the patient will not regain consciousness. Although this is a general rule, two patients made complete recoveries, without signs of cerebral damage, after their hearts had stopped four times during operation.

Widely dilated pupils may be indicative of cerebral accident. They may, of course, indicate simply a reaction to atropine. Careful observation of the pupils should be made before, and frequently during, anesthesia. Note is taken of the equality of the pupils before anesthesia. If either is larger, that fact should be recorded on the anesthesia record. A number of patients have developed Horner's syndrome during the exploration and preparation of the systemic vessel.

Because we have not found it necessary and prefer not to mask any sign of cerebral accident, curare has not been administered to any of these patients. There would not seem to be any contraindication to its use. When cerebral thrombosis occurs, the respirations may become shallow, rapid, and gasping in character. Increasing the depth of anesthesia has not been followed by notable improvement in respirations if the cause is cerebral.

Convulsions have been observed in several patients. The common cause has been heat retention or carbon dioxide excess. During the hot weather, the canister is supported on an ice cap. We believe that this measure has afforded considerable benefit. The canister is changed as frequently as necessary and at least every hour. If the convulsions are believed to be of cerebral origin, the anesthesia is maintained in the lightest possible plane. Hyperpyrexia as a plasma reaction has been observed in a few cases.

Pulmonary edema arising during or immediately after operation has not been encountered in this series. Considerable mediastinal emphysema was seen in the early cases but is rarely noted today. We may be employing slightly lower positive pressures, but there has been little essential change in the management of anesthesia for this second series of cases.

Intubation of infants and small children has been believed to be dangerous. Laryngeal edema of serious degree was anticipated. However, the greater ease of control of the anesthesia and respirations with the intratracheal technic has prompted us to use it for every patient. The incidence of laryngitis or edema has been surprisingly low. Two patients underwent tracheotomy. One, a 5 year old white girl, had congenital absence of the lower half of the sternum. Inspiration caused deep retraction of the chest under normal conditions. A cerebral accident occurred during operation, and the respirations were labored. Tracheotomy was performed as a last resort without benefit. The trachea proved to be entirely unobstructed and free from secretions. This patient died twenty hours after operation without regaining consciousness. The second patient, a 2 year old white boy, developed tracheitis and high fever twenty-four hours after operation. Six hours after on-

set, a tracheotomy was performed with prompt relief of all symptoms.

Approximately 25 per cent of all patients had some croup. A steam kettle or croup tent has been used for supportive treatment.

Age or difficulty with intubation cannot be correlated with the incidence of laryngeal complications. A large percentage of the infant group had no respiratory difficulties. Conversely, several patients in the older age group developed croup after easy intubation.

Three patients came to operation twice. Each of these patients reacted poorly to the anesthesia or the operation the first time. The operation was not begun on the first patient and was discontinued before the anastomosis was started on the remaining two patients. These cases are reported in some detail.

Case 340.—A 10 year old white boy developed auricular tachycardia immediately after intubation had been accomplished under cyclopropane-ether-oxygen anesthesia. Pressure on the carotid sinus did not reduce the pulse rate which was recorded at 300 on the electrocardiograph. Lanatosid C (Cedilanid) given intravenously also had no immediate effect, and it was deemed inadvisable to operate. This boy was returned to the ward and maintained on digitalis for ten days. A second attempt to operate was then made. No complications of any nature were encountered at this operation, and the child made a rapid recovery with an excellent result.

Case 363.—A 5 year old white girl developed repeated slowing of the heart immediately after the pleural cavity was opened. Prior to this slowing, the anesthetic had been cyclopropane-ether-oxygen. Ether-oxygen was the sole anesthetic agent after the first incident. The heart responded promptly to atropine given intravenously, but slowed again when the lung was gently retracted to expose the mediastinal vessels. Inflation of the lung with oxygen brought about a prompt increase in the heart rate. Bradycardia was the immediate response to two additional attempts to expose the pulmonary artery. Operation was abandoned, and the chest was closed with no further complications. This child was digitalized, and fifteen days later a completely successful anastomosis was performed without any untoward reactions. Her recovery was uneventful and the result of operation excellent.

The anesthesia was conducted with the same agents and by the same technics at both operations for these two patients. There appears to be a group of patients who will withstand surgery better after adequate digitalization.[11]

Case 501.—A 9 year old white girl had been pursuing a steadily downward course at home. When she came to operation, she was in mild cardiac failure. Induction of anesthesia was accomplished with cyclopropane-ether-oxygen followed by ether-oxygen after intubation. It was not possible to determine the blood pressure by auscultation before anesthesia. It

11. Ibid.

was 90/? by palpation and fell to 70/? after intubation. Shortly thereafter, during the clean-up period, the blood pressure could not be determined. The pulse was weak and thready, and the rate varied from 80 to 100 per minute. d-Desoxyephedrine hydrochloride (Drinalfa), 5 mg., was administered intravenously, and the blood pressure was restored to 100/? The pulse continued to be variable in quality, ranging in rate from 100 to 120 per minute. Spreading of the rib retractor caused marked slowing of the heart and rapid jerking respirations. Release of the retractor and inflation of the lung with oxygen, plus the administration of atropine intravenously, improved the cardiac tonus and activity. Two additional attempts were made to expose the pulmonary artery with identical reactions. The pupils were now moderately dilated, and it was feared that the patient had suffered a cerebral accident as a result of the comparatively long period of hypotension and the three episodes of weak cardiac action. Operation was discontinued without exposing the artery. She was returned to her bed in poor condition and without signs of returning consciousness. She did regain consciousness two hours postoperatively and made a slow recovery from the operation.

Since operation was deemed this child's only chance for survival, a second attempt to perform an anastomosis was made thirteen days after the first operation. She was well digitalized in the interim. The anesthesia was conducted in the same manner as at the first operation. As a prophylactic measure, atropine, 0.2 mg., was administered intravenously when the pleura was opened, and the exposed vagus nerve was injected with 1 cc. of 2 per cent procaine. During performance of the anastomosis, slight arrhythmia developed, and lanatosid C (Cedilanid), 0.4 mg., was administered intravenously. The right temporal pulse was markedly stronger than the left during operation. Persistently rapid respiration, 32 to 40 per minute, was the only disturbing complication of this second operation. There was great improvement in her color after the anastomosis was opened, and she was returned to the ward in satisfactory condition. The immediate postoperative course was encouraging, but thirty hours later she developed cardiac decompensation and died of pulmonary edema and cardiac failure on the third day after operation.

Two patients died during the performance of the anastomosis. In one case, the heart slowed to complete arrest three minutes after the occluding clamp had been placed on the pulmonary artery. Cardiac arrest occurred in the second patient twelve minutes after the occluding clamp had been placed. All efforts to restore cardiac activity were futile in each case. Autopsy revealed a single pulmonary artery in both instances. These two accidents led to the routine occlusion of the pulmonary artery, for five to ten minutes, as a test measure, before starting construction of the anastomosis. This is usually

done during exploration of the systemic vessels. The likelihood of occluding a single pulmonary artery has been lessened by angiocardiographic studies, which are now made in all doubtful cases.

Two patients (cases 54 and 294) came to operation for a second subclavian-pulmonary artery anastomosis during the series. The first operation was performed when each child was 8 months old. Only slight benefit had been derived from the first operation. Their vessels were small, and the increased pulmonary circulation was deemed inadequate. At the ages of 4 years and 15 months respectively, a second anastomosis was made on the opposite side with complete success.

Deaths

Twenty-two patients died during anesthesia or surgery. Five of these deaths occurred suddenly during chest closure. Of these five, one patient was in very poor condition during the operation. Her heart had slowed to arrest four times during the procedure. The first episode came without warning after the anastomosis had been started. The second occurred before the anastomosis had been completed. A third arrest came during the chest closure. The chest was immediately reopened and the heart action restored. A second closure of the chest was made rapidly, but two minutes after the operation had been completed the heart action ceased for the fourth and last time. All efforts to revive it were unsuccessful. Autopsy revealed that this patient did not have tetralogy of Fallot but did have gross abnormalities of the heart valves.

No explanation could be found for the fatal outcome in the remaining four cases. In each case the anesthesia had been discontinued and oxygen-helium or oxygen-air had been administered for at least ten minutes before cardiac arrest occurred. Each patient was deemed to be in good condition and had shown marked improvement in color. Each suddenly became pallid with an imperceptible pulse and blood pressure. In each case the chest was immediately reopened and cardiac massage instituted, plus the administration of stimulants and continuous artificial respiration with oxygen. No response was obtained.

We have been at a loss to explain why these deaths should have occurred at this stage in the procedure. It is possible that the positive pressures employed were excessive and that alveolar ventilation was inadequate.

Eleven patients died within twenty-four hours of operation from various causes. The causative factors in the fatalities have not always been revealed at autopsy. In all probability the anesthesia has been a contributing factor in some cases. The anesthetic agents and technics of administration are essentially the same for all patients, but there is a wide variation in cardiac irritability and reserve in these patients, and anoxia of the myocardium cannot be tolerated by a certain percentage of them.

Postoperative Course

Common postoperative complications which may occur are: cardiac decompensation, pulmonary edema, cerebral thrombosis, pneumothorax, pleural effusion, phlebitis, laryngeal edema, and traumatic injuries.[12] The usual convalescence is from two to three weeks. A marked improvement in color may be noted during this period, as well as a considerable increase in exercise tolerance. Personality changes are often striking.

Summary Of Cases

There have been 480 anesthesias administered to 475 patients in this series. In 41 cases the anesthetics were administered by physician anesthetists. The remainder were administered by six nurse anesthetists. It has been the writer's privilege to administer 289 of these anesthetics.

The youngest patient was aged 4 months and the oldest 45 years. Fifty-four patients were over 20 years of age, and four were over 30.

The average anesthesia time has been three hours and thirty-five minutes. The longest anesthesia lasted six hours and fifteen minutes, and the shortest, one hour and forty minutes.

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Summary

A review has been made of 480 anesthesias administered to 475 patients for the surgical treatment of pulmonary stenosis. Anesthesia in each case has been administered by the intratracheal, to-and-fro absorption technic, with cyclopropane-oxygen, cyclopropane-ether-oxygen, or ether-oxygen as the anesthetic agents. Bradycardia is the most frequently encountered complication and is treated successfully with atropine given intravenously or by increased oxygen tension. The importance of visual observation of cardiac tonus and action cannot be overemphasized. Minimal mediastinal movement is provided in first plane anesthesia. Maintenance of adequate alveolar ventilation throughout operation is essential to the safety of the patient. Patients with comparatively normal hemoglobin and hematocrit findings and with high oxygen saturation, who exhibit marked intolerance for exercise, are the ones who frequently present difficulties during anesthesia.

The advice of Dr. Austin Lamont and Dr. Merel H. Harmel was invaluable during the early cases of this series. Dr. Alfred Blalock's patience and unfailing confidence in us is sincerely appreciated.

MECHANICAL CONTROL OF BREATHING DURING TRANSTHORACIC SURGERY

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It is not the aim of this paper to review the literature on the technic of anesthesia for transpleural operations, as this has been covered in a number of excellent articles. The purpose of this article is to describe a technic of anesthesia used successfully in a series of transthoracic operations at University Hospitals of Cleveland—the technic of mechanically controlled breathing and the use of curare. This series of cases includes the following types of operations: pneumonectomy, lobectomy, transthoracic vagotomy, operation for diaphragmatic hernia, esophagectomy, transpleural gastrectomy, pericardectomy, ligation of patent ductus, and Potts' operation.

Definition of Terms

Terms encountered in the literature on anesthesia for transpleural operations which concern lung ventilation include: controlled breathing; apneic technic; passive respiration; aided, supplemented, assisted, augmented, or compensated breathing; paradoxical breathing or pendulum air[7,20]; pulmonary decompensation[5]; bucking[5]; intermittent positive pressure; constant positive pressure; and positive pressure breathing. There is a lack of uniformity in the use of some of these terms which at times, unfortunately, tends to obscure the author's exact meaning. To avoid possible further misinterpretation, the terms used in this paper will be defined.

By controlled breathing, passive respiration, or apneic technic[9] is meant the deliberate production of apnea by one or several of the following physiologic and pharmacologic factors: raising the respiratory threshold to carbon dioxide stimulation by means of medication and/or the anesthetic; by hyperventilation[7,9,12]; through alteration of the activity of the Hering-Breuer vagal reflexes; administration of curare to produce relaxation, *but not paralysis* of muscles concerned in respiration. With this technic the anesthetist assumes complete control of the patient's lung ventilation by controlling rate, volume, and rhythm.

Assisted, aided, supplemented, augmented, and compensated breathing are terms used synonomously to mean that the patient's

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[NOTE: References are included in a bibliography at the end of the article.—Ed.]

own continuously spontaneous respiratory activity is guided by the anesthetist. This is accomplished by intermittent manual pressure on the breathing bag. The rate is set by the patient, the volume by the anesthetist; the rhythm may be somewhat modified by the character of the augmentation.

By intermittent positive pressure is meant pressure above atmospheric on inspiration and release of pressure to *atmospheric* pressure on expiration.

By constant positive pressure is meant pressure above atmospheric on both phases of the respiratory cycle, varying or being varied on each phase but being maintained above atmospheric pressure at the end of expiration.

Anesthetic Agents and Technics

Not only is there lack of uniformity in the use of terms to describe the various technics of anesthesia for transpleural operations, but also lack of agreement among outstanding anesthetists and surgeons in regard to the anesthetics and the technics for this type of surgery.[1,2,3,4,5,-7,17,19,21,22,23]

Nitrous oxide and oxygen; nitrous oxide in combination with pentothal sodium and curare; avertin in combination with other agents; ethylene; cyclopropane; ether; and spinal anesthesia have all been advocated. Constant positive pressure breathing with augmentation, constant positive pressure breathing without augmentation, intermittent positive pressure (augmented breathing), controlled breathing (apneic technic), and no augmentation and no positive pressure—each has been suggested by various writers as the preferred technic.

It would seem, however, that all are in agreement on the basic requirements for anesthesia in transthoracic surgery. These are:

1. A completely patent airway
2. Maintenance of adequate pulmonary ventilation
3. Minimal interference with pulmonary circulation
4. Prevention of noxious respiratory and circulatory reflexes
5. Stabilization and prevention of tearing of mediastinum
6. A quiet operative field
7. Prevention of aspiration of secretions into the sound lung
8. Prevention of atelectasis
9. Prompt return to consciousness

The Mechanical Respirator

Mechanical control of breathing during anesthesia was reported by Crafoord of Sweden in 1938.[7] Aside from Crafoord's work the only published material on mechanical control of breathing during anesthesia found by this writer is by Mautz of Western Reserve University,[12,13] designer of the Mautz respirator (the one used in this clinic) (fig. 1), and by Mautz, et al.[14]

This mechanical respirator is a simple device that can be fitted with attachments for use with any gas machine with a carbon dioxide absorber of either the circle or to-and-fro type. The respirator consists of a breathing

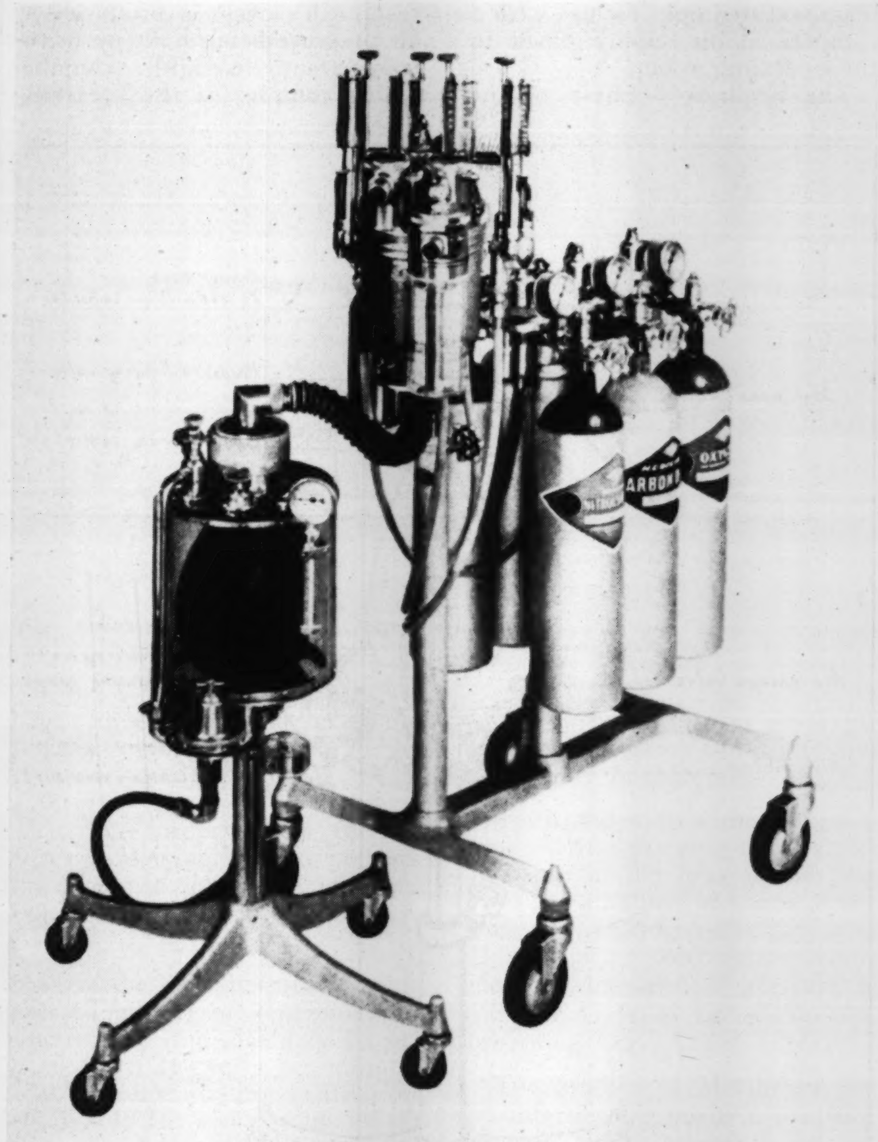

Fig. 1.—The Mautz respirator attached to Heidbrink Kinetometer anesthesia machine.

bag enclosed in a transparent airtight chamber; a windshield wiper-like mechanism attached to both a respiratory rate control valve and an air egress valve; a pressure limiting valve; and an air ingress tube (fig. 2). At the present time the respirator is

manufactured only for use with a compressed air supply piped to the operating room.

The inspiratory phase of the respiratory cycle is produced by air pressure being built up in the transparent airtight chamber which compresses the breathing

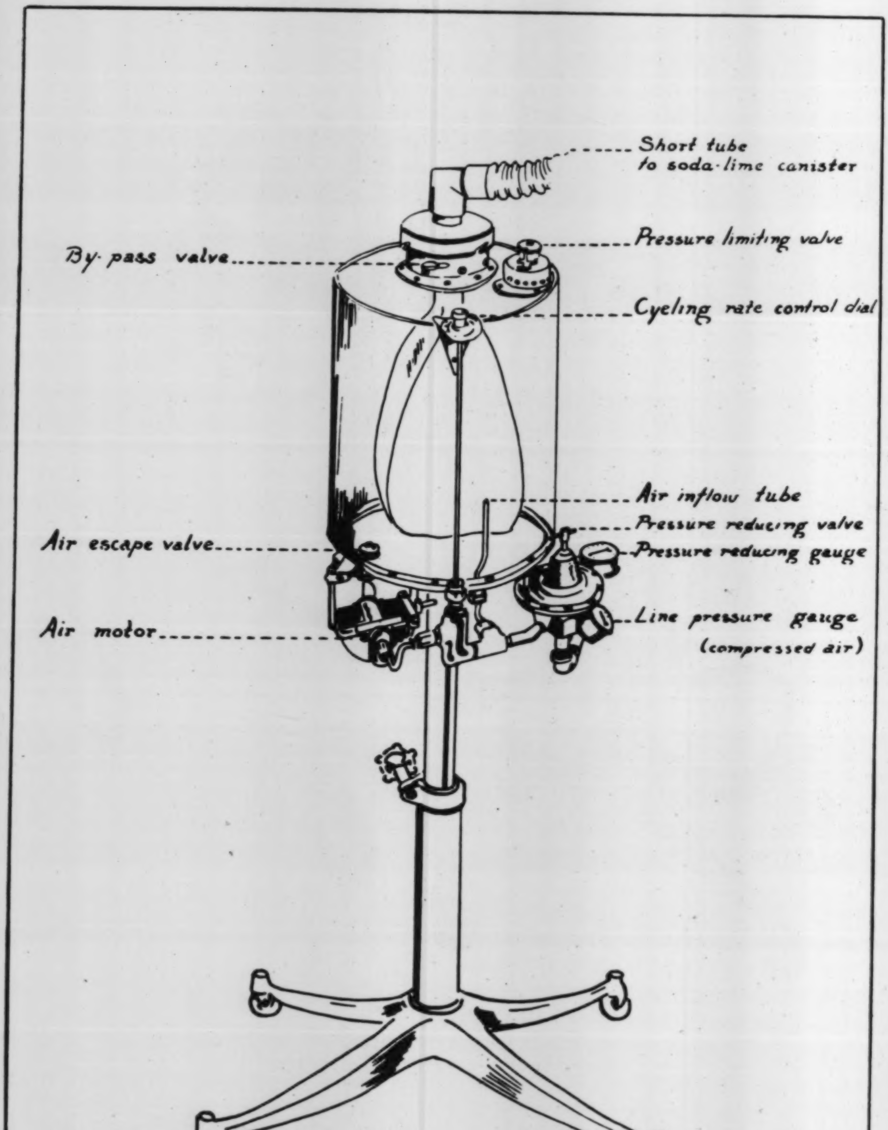

Fig. 2.—Diagrammatic sketch of Mautz respirator.

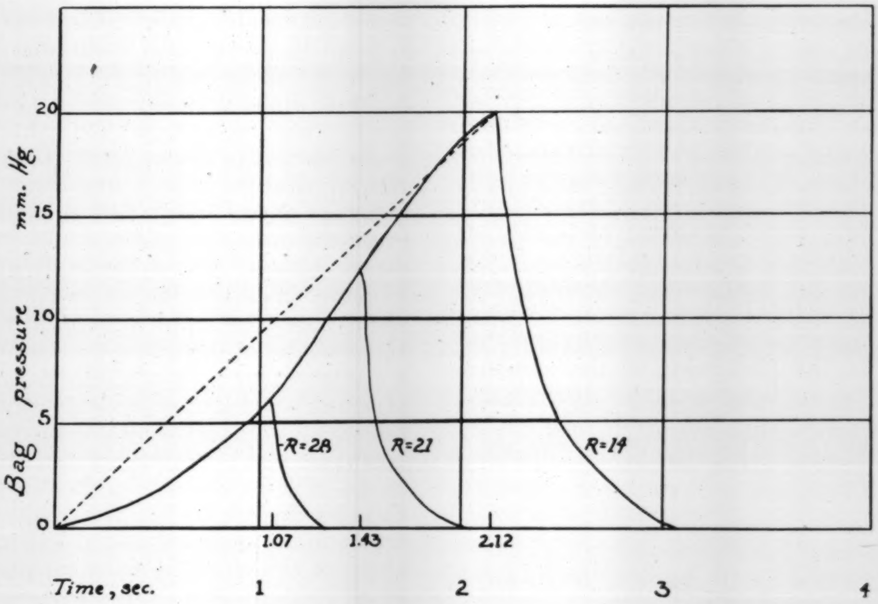

Fig. 3.—Variations of time-pressure relationships with the Mautz respirator with compressed air pressure set at 4 pounds per square inch.

bag. Pressure in excess of the set pressure escapes through the pressure limiting valve. The expiratory phase of the respiratory cycle occurs when the air egress valve opens and permits the pressure in the chamber to fall rapidly to atmospheric pressure.

With the line pressure of compressed air set at 4 pounds per square inch, the rate may be set from 14 to 24 cycles per minute. The pressure of lung ventilation obtainable (to a maximum of 20 mm. Hg) is in proportion to the amount of compressed air that enters the chamber with the various rates of cycling at this set pressure (fig. 3). But by varying (1) the rate of inflow of compressed air into the chamber, (2) the cycling rate, and (3) the lung ventilation pressure, a great variety of pressure-time relationships may be obtained.

With the use of the intermittent positive pressure technic, the pressure in the lung cannot exceed the maximal pressure of the pressure limiting valve (20 mm. Hg). But pressure relationships may be deranged if pressure is allowed to build up within the breathing bag.

The studies of Motley et al[15] on intermittent positive pressure showed that there was no decrease in cardiac output with ventilation that built up pressure gradually during inspiration with a rapid decrease of pressure early in expiration to atmospheric pressure and an expiratory time equal to or longer than inspira-

tory time. The operation of the Mautz respirator fulfills these requirements.

The advantages of mechanically controlled ventilation over hand or hand-knee pressure control are: precision of cycling rate and time-pressure relationships; elimination of the fatigue factor on the part of the anesthetist as it relates both to continuous management of lung ventilation and to the well-being of the anesthetist on long operative procedures; and freeing the anesthetist's hand from the breathing bag for other duties.

Anesthetic and Technic

The adult patient is given a barbiturate the night before he is scheduled for operation, and morphine sulfate, 0.010 to 0.015 Gm., and atropine sulfate, 0.00045 to 0.0006 Gm., are administered about one hour before operation. Premedication for children is administered proportionally according to age, size, and physical status. Normal saline solution administered intravenously by cannula or needle is started prior to the operation. After the operative procedure has begun, whole blood is administered. The estimated amount of blood loss and the condition of the patient's circulation (blood pressure, pulse rate, and volume) are the criteria for the determination of the amount of blood and the rate at which it is given.

The closed carbon dioxide absorption technic is used routinely. Nitrous oxide-oxygen by the semi-closed technic is used for induction. The breathing circuit is then closed, the nitrous oxide shut off, and ether added as rapidly as the patient will tolerate it. No additional nitrous oxide is added unless the breathing circuit is not leak-proof. Anesthesia is carried to lower plane 2. A Magill tube to which has been attached an inflatable cuff is then inserted into the trachea under direct vision. Sizes 38 and 40 French tubes are the most commonly used for adults. A mouth bite, made of rolled gauze and slightly larger than the diameter of the tube, is immediately placed between the teeth. The intratracheal tube is connected directly to the to-and-fro soda lime canister by means of the canister-catheter adapter or to the tubes of the circle filter by means of a Y connection. This technic is used in order to prevent distention of the stomach which may occur when intermittent positive pressure is used with the face mask technic. The patient's respiratory activity is very carefully evaluated for complete patency of the airway.

When the patient is placed in the lateral position, no sandbag or rest is placed under the dependent side. The arm on the operated side may be fastened to the anesthetist's screen or supported on an armboard. When the patient is in the lateral position, an effort is made to keep the weight of the arm off the upper chest.

Anesthesia is maintained in mid-plane 2 prior to the opening of the pleura. The respirator is attached to the gas machine during the time of approach to the pleural cavity but is not put into operation. When the pleura is

opened, the respirator (set at about 10 mm. Hg pressure) is put into operation. One-half cc. (10 units) of curare (Introcostrin) is administered intravenously. Usually this amount of curare is sufficient to establish control of respirations. If control is not established within two or three minutes, an additional 0.5 cc. curare is administered intravenously, and as soon as control is established the ether is discontinued. The operative field is kept under close observation by the anesthetist, and the amount of pressure used is determined by the degree of lung inflation. The usual amount of pressure required for the inspiratory phase ranges between 5 and 10 mm. Hg. After control is established, the cycling rate, in the majority of cases, is maintained between 14 and 18 per minute. The diaphragm maintains slight activity, and the abdominal muscles are inactive.

The diaphragm and abdominal muscles are watched closely for increasing activity. As diaphragmatic activity increases and abdominal movement begins, slight movement of the breathing bag, independent of the cycling rate, will be noted. At this point ether is again turned into the circuit, and the diaphragmatic activity will diminish. When no intercycling movement is noted in the breathing bag, the ether is again cut off. The next time this slight spontaneous respiratory activity is noted, an additional 0.5 cc. curare is given. By alternating the administration of ether and small doses of curare as outlined, the patient can be maintained in control but will resume spontaneous respirations if *rhythmic* intermittent positive pressure is discontinued. This has been proved repeatedly during suctioning of the trachea, and when it is desired to allow the patient to resume spontaneous respiratory activity at the close of the procedure. After control is established, the mediastinum is stable, and there is very slight movement of the diaphragm and no abdominal movement. The lung on the open side expands on inspiration and deflates on expiration. When the expanding lung interferes with the work of the surgeon, it is gently retracted. Whenever change in lung volume is made by the surgeon, adjustments are made in the volume of gas in the breathing bag so that the pressure at the end of each expiration falls to *atmospheric.* Whenever packs or retractors are released, the area of the lung against which they were pressed usually re-expands without change in pressures, but if it does not, slight additional pressure will produce re-expansion. Re-expansion of resistant lung areas is always done gently so that no injury will be done by the increased pressure to the already expanded alveoli.

Although a leak-proof system has been established at the beginning of the operation, leaks may develop after the operation is under way. This may occur during the use of intermittent positive pressure, or the gases may leak out through an area of the lung being operated upon, or the air in the inflated cuff may leak out. Whenever necessary, the

flow of gases from the anesthesia machine is increased to compensate for leaks.

The trachea is aspirated during the operative procedure as indicated, with a 14 or 16 French, 20 inch, two-hole rubber catheter. If a local anesthetic has not been injected around the hilus, anesthesia is deepened during any manipulation around these structures to diminish vagal reflexes. An additional dose of atropine may be indicated to diminish vagal response to stimuli.

No curare is given after the closure is begun, and it is seldom necessary to give any ether after this time. If the patient is still controlled, the use of the respirator is continued. If respiratory activity is resumed by the patient the respirator is disconnected, the rebreathing bag attached to the canister, and augmented breathing, using intermittent positive pressure, is carried out. Before closure of the pleura is started, the lung is fully expanded and then partially deflated so that no injury may occur to the visceral pleura during closure of the parietal pleura. Before the seal of the pleural cavity is effected, the lung is again fully expanded, and continuous positive pressure may be used for several minutes at this time to prevent air from being trapped in the pleural cavity.

If the respirations are still controlled and the respirator is still in use when the pleural seal is made, the respirator is then disconnected and the patient allowed to resume spontaneous breathing. Resumption of spontaneous breathing is accomplished by waiting three or four seconds, then inflating the lungs by manual pressure on the breathing bag to insure adequate oxygenation; again waiting three or four seconds, then inflating the lungs. After a few repetitions of this procedure, the patient's respiratory activity will be noted. The anesthetist then synchronizes the pressure on the breathing bag with the patient's inspiratory movement on every third or fourth breath. This is repeated as long as respiratory activity is inadequate. By the time closure of the wound is completed, it is rarely necessary to augment breathing. During the last few minutes of the operative procedure, helium 70 per cent and oxygen 30 per cent is administered as a prophylactic measure against atelectasis.

Postoperative Care

The trachea is aspirated thoroughly through the intratracheal tube; then with the suction catheter inserted through the intratracheal tube (the end of suction catheter lower than the end of the intratracheal tube) both tubes are withdrawn. Further tracheal suctioning is done as indicated. In certain cases bronchoscopy is done.

The chest may be closed with or without constant drainage of the pleural cavity. If closed without suction, the chest is examined, and if air has been trapped in the pleural cavity, it is aspirated. If full intercostal activity has not returned, the patient is kept in the anesthetizing room adjacent to the operat-

TABLE 1.—ANESTHESIA RECORD IN 64 CASES IN WHICH ETHER, CURARE, AND MECHANICALLY CONTROLLED BREATHING WERE EMPLOYED

OPERATION	DIAGNOSIS	NO. CASES	AVER. AGE-YRS.	AVERAGE TIME ANESTHESIA HOURS	AVERAGE TIME ON RESPIRATOR HOURS	AVERAGE TIME RECOVERY TIME	CURARE AVER. DOSE CC.
Resection of stomach	Carcinoma	3	55	3½	1¾	45″	2
Esophagectomy and gastrectomy	Carcinoma	6	56.8	4¼	2¾	29″	3
Esophagectomy.	Carcinoma	3	50.6	4¼	2	30″	4
Vagotomy	Peptic ulcer	13	37.1	2¾	2	48″	3.2
Diaphragmatic herniotomy	Hiatus hernia	5	57.5	3¼	1½	1° 16″	2
Lobectomy	Bronchiectasis	5	45.1	4¼	3	31″	2.4
Pneumonectomy	Carcinoma lung	10	55	4¼	3	1° 53″	3.1
Exploratory thoracotomy	Carcinoma (2) Traumatic defect (1)	3	50	2½	¾	30″	2.3
Pericardectomy	Pericardial scar	2	36	4¾	4	40″	5
Ligation patent ductus	Patent ductus arteriosus	9	11.3	3½	2½	45″	2.2
Potts' operation Exploration pulmonary artery	Tetralogy of Fallot (2) Essenmenges syndrome (1)	3	6.7	4¾	3¼	52″	3
Biopsy mediastinal node Mediastinal exploration and excision dermoid cyst	Carcinoma lung (1) Dermoid cyst (1)	2	36	2¾	1½	25″	2

ing room until chest expansion is satisfactory. Oxygen (preferably by nasal catheter) is administered. As soon as full intercostal activity has returned, the patient is taken to the division by the anesthetist with oxygen being administered by nasal catheter in transit. Postoperatively, oxygen by tent or catheter is administered. Tracheal suction is done as needed. An x-ray of the chest is taken, and in the majority of instances the patients are gotten out of bed the day after operation.

Summary of Results

Results of 64 cases in which ether, curare, and mechanically controlled breathing were employed are listed in table 1 and table 2. The cases are presented according to type of operation, and in a few instances operations of similar type have been grouped together.

The youngest patient in this series was 4 years old; the oldest was 70. The average duration of anesthesia for each group ranged from 2 hours and 15 minutes to 4 hours and 42 minutes. The average time during which mechanically controlled breathing was employed for each group ranged from 47 minutes to 4 hours. The average time of recovery from anesthesia for each group (as accurately as this can be known from recorded data on nurses' bedside notes) ranged from 25 minutes to 1 hour and 53 minutes. The latter was for the pneumonectomy group. The average recovery time from anesthesia for the total number in the series was 46 minutes. The total average dose of curare for each group ranged from 2 cc. to 5 cc. (table 1).

The anesthesias in this series were essentially uncomplicated. One patient developed marked but transient circulatory disturbance when the ribs were spread, and two patients developed circulatory disturbance during operative manipulations around the hilar structures. There was no specific pattern of circulatory changes which seemed to be attributable to mechanically controlled breathing, and any abnormal circulatory changes which occurred appeared to be on the basis of the operative procedure rather than on the technic of anesthesia.

Table 2 shows the postoperative complications, deaths, and causes of death. There were six deaths in this series. The earliest death occurred on the third postoperative day, the latest on the seventeenth postoperative day. According to the records no deaths in this series were attributable to the anesthesia.

Comments

In regard to controlled breathing, Beecher[1] has stated: "With the respiration eliminated, the anesthetist's first real warning of serious overdosage comes only at about the time signs of circulatory failure are established." Our experience with controlled breathing has not borne out this statement. In our series of cases with controlled breathing, overdosage has not occurred, and the majority of the patients who have had curare are reacting at the completion of the procedure and

TABLE 2.—POSTOPERATIVE COMPLICATIONS AND DEATHS IN 64 CASES IN WHICH ETHER, CURARE, AND MECHANICALLY CONTROLLED BREATHING WERE EMPLOYED

OPERATION	NO. CASES	DIAGNOSIS	DEATHS			OTHER COMPLICATIONS	
			NO.	CAUSE	NO. DAYS POST-OPERATIVE	TYPE	NO.
Resection stomach	3	Carcinoma					
Esophagectomy and gastrectomy	6	Carcinoma	2	Mediastinitis Metastasis	4th 9th		
Esophagectomy	3	Carcinoma				Cough Pleurisy	1 1
Vagotomy	13					Patchy atelectasis Pneumothorax	3 1
Diaphragmatic herniotomy	5	Hiatus hernia				Pleural effusion Possible embolus and possible coronary	1 1
Lobectomy	5	Bronchiectasis				Emphysema Pneumothorax Empyema	1 1 1
Pneumonectomy	10	Carcinoma	2	Pericarditis and pulmonary embolism Hemothorax	12th 17th	Hydropneumothorax	2
Exploratory thoracotomy	3	Carcinoma (2) Traumatic defect (1)	1	Inoperable carcinoma	3rd	Pleural effusion	1
Pericardectomy	2	Pericardial scar	1	Hemothorax, hydrothorax, and patchy atelectasis	4th		
Ligation patent ductus	9	Patent ductus arteriosus				Laryngeal edema Pneumonia Atelectasis	1 1 1
Potts' operation	2	Tetralogy of Fallot				Pleural effusion	2
Exploration pulmonary artery	1	Essenmenges syndrome				Patchy atelectasis	1
Biopsy mediastinal node	1	Carcinoma lung				Atelectasis	1
Mediastinal exploration and excision dermoid cyst	1	Dermoid cyst					

can be returned to the division promptly. We have been using curare for about one year, and controlled breathing with nitrous oxide-oxygen-ether has been used for a much longer period.[14]

Another objection to the use of control of breathing is the reduction of arterial carbon dioxide below normal levels and the production of alkalosis. Guedel[9] stated in his original article on ether apneas, "The question is raised as to the effect upon the patient of the lowered blood carbon dioxide in the maintenance of these apnoeas. The answer is, that the postoperative patient does not show an alkalosis of carbon dioxide depletion. The disturbance of the acid base equilibrium is far less than with the usual open ether anesthesia." Although no blood studies have been carried out on these cases, spontaneous respiration is resumed within a matter of seconds after the respirator is cut out of the breathing circuit.

Orton[19] maintains that controlled breathing has been condemned owing to a misunderstanding of the basic principles involved. "It has been stated that hyperventilation will produce dangerous acapnea and alkalosis. But, when using controlled respiration, the object is not to produce hyperventilation and lowering of the carbon dioxide level of the blood. The aim is to maintain normal ventilation in the presence of a raised threshold and so abolish spontaneous respiration."

Nosworthy's[18] work with cyclopropane and controlled breathing technic showed adequate ventilation and no reduction of carbon dioxide below normal.

In a final consideration of the various technics of anesthesia in current use in transthoracic surgery, it would seem that the bulk of opinion is in favor of the technic of intermittent positive pressure which may provide adequate oxygenation, elimination of excess carbon dioxide, minimal interference with pulmonary circulation, and reduction in metabolic activity; and opposed to the technic of constant positive pressure which may provide adequate oxygenation, but permits retention of carbon dioxide, interferes to a greater extent with pulmonary circulation, and causes an increased metabolic activity.[3,4,5,6,7,8,13,18,20]

Intermittent positive pressure may be carried on with controlled or augmented breathing, and controlled breathing may be carried on by mechanical or manual means. Whether, in the future, we will see a wider use of augmented breathing or controlled breathing, and, if controlled, a wider use of mechanical or manual control, will have to depend upon evidence from further studies carried on in the clinic and in the research laboratory.

Conclusions

When the controlled breathing technic is used in transthoracic surgery during the period when the pleural cavity is open, mechanically controlled breathing appears to be advantageous since it provides precision of lung ventilation in regard to rate of cycling and time-pressure relationships. The maintenance of

precision of lung ventilation manually by the anesthetist for any period of time is impossible from the standpoint of the fatigue factor and the distraction from the task of performing manual pressure in order to take pulse and blood pressure and record data on the anesthesia record, and so forth. This factor of precision may bear a relationship to increased ease of maintenance of apnea.

In the majority of cases in the series it has been possible to establish controlled breathing with the patient in mid plane 2 anesthesia and the administration of 0.5 to 1 cc. curare; to maintain control of breathing at a level lighter than mid plane 2 with small total doses of curare; and to re-establish the patient's spontaneous respiratory activity rapidly when desired. Further studies on the production and control of apnea would seem to be indicated. Perhaps this might be on the basis of reduction in metabolic activity during passive respiration and on the basis of other reflexes of respiration rather than solely on production of acapnia with a respiratory threshold raised to carbon dioxide stimulation and a high concentration of oxygen.

The results in this series of transpleural operations with the use of an intratracheal tube, ether, curare, and mechanically controlled breathing as described have proved satisfactory from the standpoint of:

1. The patient's reaction to this technic both during the operative procedure and postoperatively.
2. Providing what appears, after clinical evaluation and comparison with other technics, to be optimal working conditions for the surgeon.
3. Precision of management of lung ventilation by the anesthetist.
4. Elimination of the fatigue factor in management when controlled breathing technic is employed and freedom of the anesthetist to take care of necessary details incident to the anesthesia and the operative procedure.

SUMMARY

Terms used to describe technics and complications of anesthesia in transthoracic surgery have been enumerated, and certain terms have been defined.

The Mautz mechanical respirator has been discussed.

A technic of anesthesia using ether, curare, and mechanically controlled breathing has been presented.

A report of sixty-four cases has been presented.

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(*Continued on page* 166)

REFLEXES IN GENERAL ANESTHESIA

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INTRODUCTION

For the purposes of this article, we shall rather arbitrarily define a normal reflex as a more or less purposeful type of activity in response to an adequate stimulus (or to summated, individually inadequate stimuli), occurring without conscious volition. Without systems of such reflexes efficiently controlling the vital activities of circulation, respiration, digestion, locomotion, and endocrine secretion (at least to a degree) with a minimum of conscious direction from the cerebral cortex, man, instead of riding in airplanes at a speed approximating that of sound and grappling with the problems of atomic energy, would probably still have all his neural activities concerned with the basic needs of protection, food, shelter, and reproduction—if indeed he were still surviving.

Such important bodily phenomena as reflexes, then, must inevitably be woven into the total fabric of medical practice and each of its specialties. So far as the specialty of anesthesiology is concerned, it might justly be said that a real anesthesiologist is a reflexologist, if such a creature exists. It is his intimate understanding of reflex fundamentals, together with his detailed and specific knowledge of certain reflexes, that permits him to alter confidently the patient's physiologic status with the optimum of safety to the patient and of convenience to his professional colleagues.

GENERAL CONSIDERATIONS

Fundamentally, most reflexes can be stripped down anatomically to the following bare essentials of the reflex arc. (Figure 1 illustrates a simple reflex arc—the stimulus, painful; the response, striated muscle contraction.[1,2,3,4]) The reflex arc consists of the *stimulus*, which must be some external or internal environmental change or changes of a kind and intensity sufficient to cause the *receptor* endorgan of the *dendrite* to initiate the *nerve impulse*. This nerve impulse propagates itself over the dendrite to the *cell body* of the *afferent neuron*, and then along its *axon* into the *central nervous system* (*brain* or *spinal cord*). Here the nerve impulse, by

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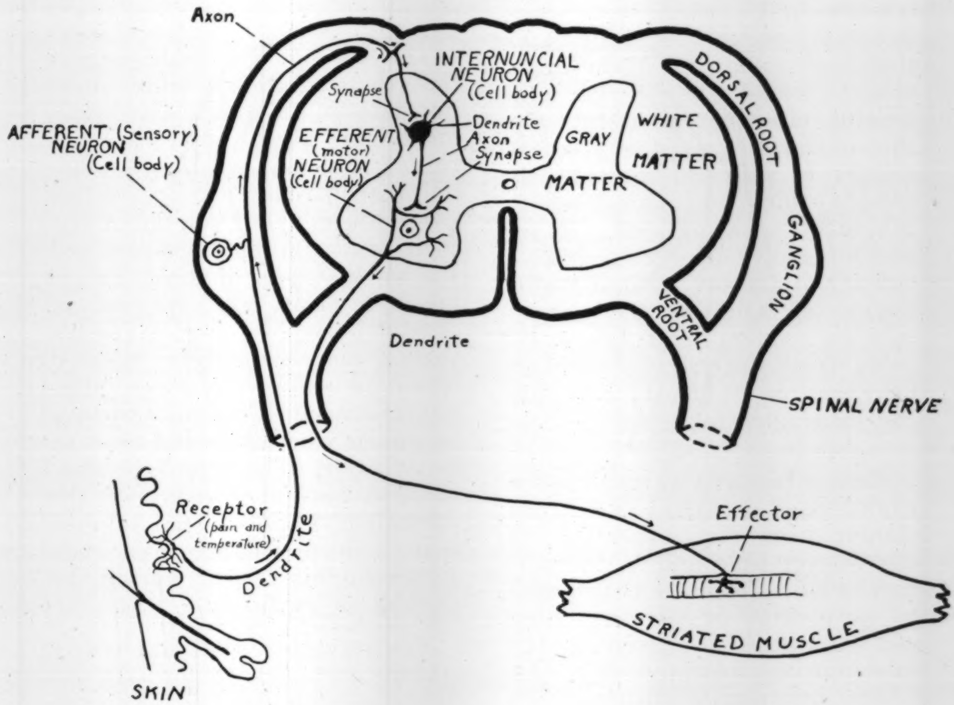


Fig. 1.—The reflex arc. Refer to text for explanation.

way of a *synapse* and after an appreciable synaptic delay, spreads to either the dendrite or cell body of the *internuncial* neuron. The latter, by way of its branching axon and another synapse, transmits the nerve impulse to the dendrite or cell body of the *efferent neuron.* Propagated over the axon of the efferent neuron, the nerve impulse reaches the *effector* ending, which may be related to the striated muscle fibers making up the motor unit, to smooth muscle or gland cells, or to heart muscle. (In the last three instances, another synapse and another neuron located in a visceral ganglion must be interposed). The nerve impulse, on reaching the effector ending, causes the aforementioned muscle and gland structures to modify their activity, the *response.*

Useful as such a basic concept of the reflex arc may be, it is obviously so highly oversimplified that error is implied at almost every turn. The diagrammatic representation of this basic concept (fig. 1), with some modification, could become a fairly accurate portrayal of an *intrasegmental* type of reflex arc—meaning by that, one which is entirely confined to a segment of the spinal cord (or brain stem) and its functionally associated receptors

and effectors. Although such intrasegmental reflex arcs undoubtedly occur, it is difficult to think of many of them that, alone, would be very purposeful, effective, or useful. On the contrary, if asked to give examples of reflexes, most of us would think of swallowing, coughing, sneezing, etc., all of which involve the co-operative activity of neurons located in different segments of the central nervous system, and hence involve *intersegmental* types of reflex arcs in contrast with the one diagramed in figure 1.

Once reflex activity involves the co-ordinated discharge of neurons distant from each other but at the correct time and in the correct numbers to make that activity purposeful or effective, then our attention is focused on the internuncial neurons (fig. 1). These internuncial neurons are the ones concerned with transferring the necessary nerve impulses about within the central nervous system. They direct the nerve impulses cephalad, caudad, or across the midline. One moment they may cause these impulses to wax in number and distribution, and the next moment, perhaps, to wane. Nevertheless, they usually direct the correct number of nerve impulses to the correct motor neurons, variously located, at just the right instant so that purposeful activity results. The more complex the reflex activity (that is, the more motor neurons involved at different levels of the central nervous system and at different time intervals in the production of that activity) presumably the more internuncials would be necessary.

The foregoing would suggest that the addition of more internuncials in the reflex arc would make for more precisely controlled, and thus potentially more purposeful, reflex activity. When we recall that they can only exert their action through synapses and that each synapse involves an appreciable delay in the conduction of the nerve impulse, it seems that a point would be reached when the addition of more internuncials would slow the response to a point where it would cease to be effective. The relatively stereotyped nature of many of the protective reflexes would suggest that a nice balance has been struck in this respect during the process of evolution.

The receptors, the sites where stimuli can act to initiate reflexes, are found primarily in the skin (pain, temperature, tactile—collectively *exteroceptors*), in striated muscle, tendon, and about joints (muscle sense—*proprioceptors*), and in relation to the viscera (*interoceptors*). Proprioceptors and exteroceptors mediate body wall and extremity, or *somatic*, sensibility; interoceptors mediate *visceral* sensibility. In addition, there are the specialized receptors for smell, vision, taste, hearing, and equilibrium.

The effector endings, the sites where reflex responses occur, are located within striated muscle fibers (the muscle being classified either as *somatic*—derived from somites—or as *branchiomeric*—derived from branchial arches), where they are called *motor end plates*. Effector endings are also

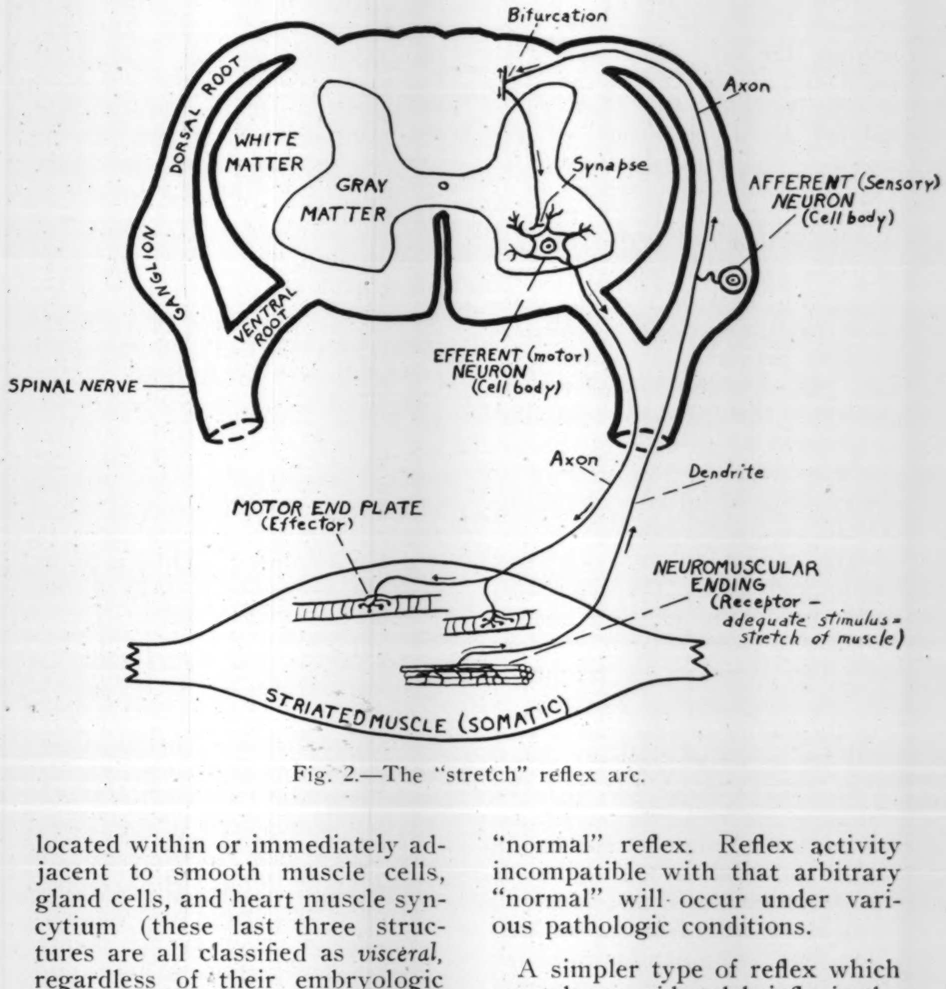


Fig. 2.—The "stretch" reflex arc.

located within or immediately adjacent to smooth muscle cells, gland cells, and heart muscle syncytium (these last three structures are all classified as *visceral*, regardless of their embryologic derivation), and are collectively called *visceral effector endings*.

Nerve impulses initiated in exteroceptive, proprioceptive, or interoceptive receptors, upon reaching the central nervous system and with the help of the appropriate internuncials, could presumably modify the activity of any type or combination of types of effectors, and would be likely to do so within the bounds of a "normal" reflex. Reflex activity incompatible with that arbitrary "normal" will occur under various pathologic conditions.

A simpler type of reflex which must be considered briefly is the "stretch reflex," since it is considered to be concerned with muscle tone.[3,4,5,6] Figure 2 illustrates this reflex arc and shows the characteristic lack of an internuncial neuron in the circuit.

3. Ranson, loc. cit.
4. Howell, loc. cit.
5. Macleod, J. J. R.: *Physiology in Modern Medicine*, 9th ed. (St. Louis: The C. V. Mosby Company, 1941).
6. Best, C. H., and Taylor, N. B.: *The Physiological Basis of Medical Practice*, 4th ed. (Baltimore: The Williams and Wilkins Co., 1946).

Here it is well to note that the efferent or *final common path* neuron, the one which sends its axon out of the central nervous system towards the effector, is subject to a multitude of demands.[3,4,5,6] Literally hundreds or even thousands of synapses occur in relation to its branching dendrites and cell body. Certain of the nerve impulses thus brought to bear on the final common path neuron are *inhibitory* in their action (i.e., decrease its rate of initiation of nerve impulses — "firing" — or completely prevent its firing); others are *excitatory* (cause the neuron to "fire," that is, initiate impulses). These two different sets of impulses appear to sum algebraically, and when the excitatory type predominates, the neuron will fire to its associated effectors, and vice versa. An important factor determining whether or not the final common path neuron will fire is its physiologic condition. If that condition is such that the neuron will fire when the excitatory stimuli streaming in are preponderant, it is said to be in a *central excitatory state;* if not, in a *central inhibitory state.* One very prominent normal factor governing the central excitatory state is how recently the neuron has fired. After firing, any neuron passes through an appreciable period when even intense excitatory stimuli applied to it cannot cause it to fire again (the *absolute refractory period*). After that, there is a period when only stimuli stronger than normal can cause it to fire (the *relative refractory period*). Not until these periods have elapsed does the neuron again approximate the normal state. These periods can be measured, but are only a few thousandths of a second in duration.

Although my opening statement excluded conscious volition as a factor in reflex activity, it must be clearly understood that conscious centers (especially the cerebral cortex) can and do modify reflex activity. Such centers act to control or to keep reflexes within bounds. By means of such conscious cortical control some types of reflex activity may be readily inhibited. This is more true of the somatic than of the visceral effector system.

So far as the relationship of general anesthesia to reflexes is concerned, it is commonly stated that general anesthetics exert their effect in an *irregularly* descending order from the higher to the lower centers of the central nervous system, phylogenetically speaking.[7,8] Thus, the cerebral cortex is first depressed, followed roughly in order by the diencephalon and striatum, the midbrain, the pons, the spinal cord, and finally the medulla oblongata. It is probably a consequence of this that the reflexes of most value to the anesthetist are those with centers in the midbrain, the pons, the medulla,

3. Fulton, loc. cit.
4. Howell, loc. cit.
5. Macleod, loc. cit.
6. Best, loc. cit.

7. Goodman, L., and Gilman, A.: *The Pharmacological Basis of Therapeutics.* (New York: The Macmillan Co., 1941).
8. Etsten, B., and Himwich, H. E.: Stages and signs of pentothal anesthesia: Physiologic basis. Anesthesiology 7:536-548, Sept., 1946.

and the spinal cord. Next to the last reflexes to be completely depressed by general anesthetics are the respiratory reflexes, with centers in the pons, medulla, and spinal cord. Last of all to be depressed are the circulatory reflexes, with centers in the medulla oblongata.

GENERAL TYPES OF REFLEXES

There are three general types of reflexes of special interest to the anesthetist. The *first* of these involves a relatively restricted change in the activity of striated muscle, smooth muscle, or glands in response to a stimulus. Typical examples are the jaw reflex, the corneal reflex, the pupillary reflexes, the laryngeal reflex, and the salivary reflex.

The *second* type is a somewhat more widespread, co-ordinated response, usually involving a more or less simultaneous change in the activity of striated muscle, smooth muscle (heart muscle), and glands in varying combinations. Swallowing, vomiting, gagging, coughing, and hiccoughing exemplify this type.

The *third* type is the group of continuously active, rhythmically integrated respiratory and circulatory reflexes. The anesthetist is vitally concerned with the constant maintenance of the reflexes of this type within a comfortable margin of safety under all circumstances.

I. Reflexes characterized by a relatively restricted response

A. Stretch reflexes, or muscle reflexes

The jaw reflex, or jaw jerk, or masseter reflex (see Wartenberg[9]), is an example of a "stretch" reflex, and its reflex arc is illustrated in figure 3. The stimulus is a rather sudden stretching of the temporal and masseter muscles by pushing the lower jaw inferiorly; the response is a brief contraction of the muscles stretched, causing the mouth to close.[10,11,12] Reference to figure 3 makes it clear that both stimulus and response are bilateral, and that no internuncial neuron is involved.

In the early stages of general anesthesia, the cortical centers have been depressed sufficiently to release the final common path neurons (fig. 3) for the jaw reflex from cortical control. Under these circumstances, the neurons of the motor nucleus of the trigeminal nerve are being bombarded by many nerve impulses set up by a variety of stimuli. Since these impulses are presented in no particularly orderly fashion, they should cause the final common path neurons to discharge asynchronously as rapidly as their various refractory periods would permit. Consequently, the masseter and temporal muscles will be in a relatively constant state of contraction during this period, with resulting clenched jaws.* At this time, of course, no jaw reflex can be obtained, since too many other stimuli are continuously monopolizing the final

9. Wartenberg, R.: *The Examination of Reflexes* (Chicago: The Year Book Publishers, Inc., 1945).

10. Flagg, P. J.: *The Art of Anesthesia,* 7th ed. (Philadelphia: J. B. Lippincott Co., 1944).

11. Guedel, A. E.: *Inhalation Anesthesia* (New York: The Macmillan Co., 1937).

12. Beecher, H. K.: *The Physiology of Anesthesia* (New York: Oxford University Press, 1940).

*This same mechanism of release from higher center control probably explains the roving eyeballs characteristic of second stage and early third stage anesthesia.

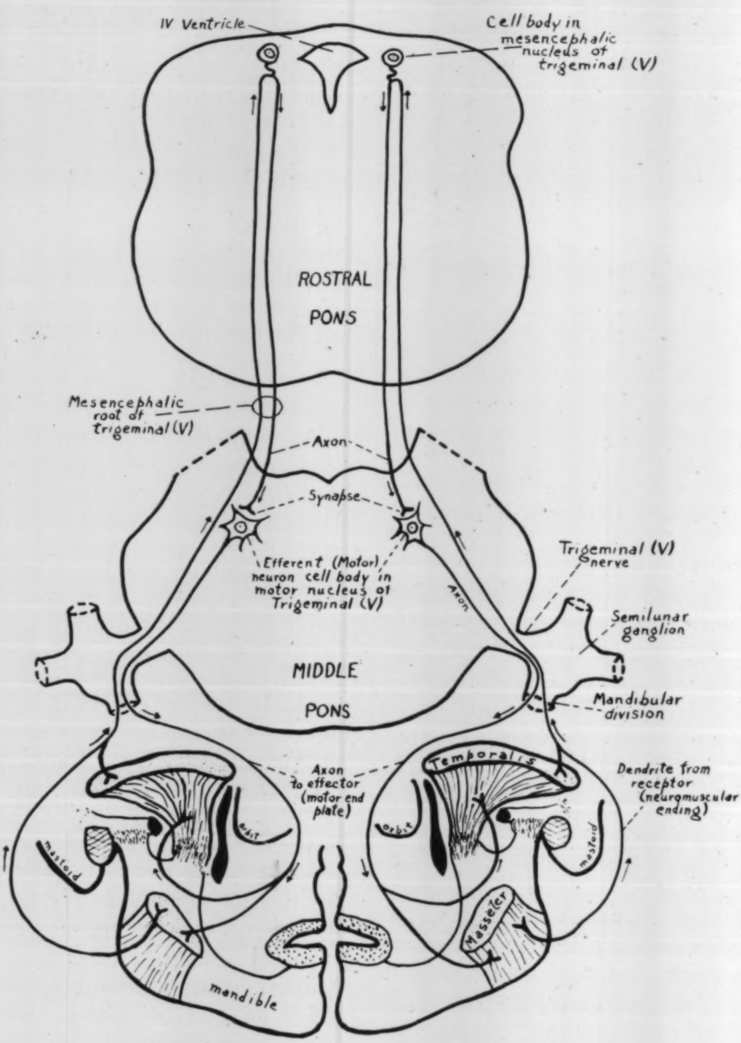


Fig. 3.—The jaw reflex arc. Refer to text for explanation.

common path neurons for this reflex. Realizing this, the anesthetist preparing a patient for intraoral surgical procedures which do not demand deep anesthesia (e.g., incision and drainage of a retropharyngeal abscess) will insert a bite block before induction.

The degree of depression of the jaw reflex, that is, the relative ease with which the anesthetist can depress the lower jaw, roughly parallels the level of general anesthesia so far as its effect on stretch reflexes elsewhere in the body is concerned. Consequently, it can be used by

the anesthetist as an indication of the degree of muscular relaxation of the patient which the surgeon *should* be experiencing, This guide is especially useful when the surgeon is working below the diaphragm, in which case it is often impossible for the anesthetist to get a direct view of proceedings. The jaw reflex is usually the last of the stretch reflexes in the head region to be depressed, and when the lower jaw can be moved about freely, the anesthetist knows that more of the anesthetic agent must be used cautiously if the respiratory reflex arcs are to be preserved (see fig. 7).[10,11,13,14] Likewise, as anesthesia lightens, either by accident or design, increasing tone in the masseter and temporal muscles will be appreciated by the anesthetist.

It must be kept in mind also, as pointed out by Flagg,[10] Guedel,[11] Lundy,[13] and others, that interference with the airway (e.g., by the presence of mucus or vomitus in the pharynx or tracheobronchial tree) may be a potent enough stimulus to keep the masseter and temporal muscles in a state of more or less tonic contraction even though the other muscles are well relaxed.[14] Consequently, in a situation in which the surgeon (especially when working in the upper abdomen or the pelvis) is satisfied with the degree of muscular relaxation and the anesthetist finds only the status of the jaw reflex inconsistent with an adequate level of anesthesia, the latter should check the condition of the airway. The anesthetist may thus be enabled to anticipate and thereby prevent threatening difficulties to the orderly maintenance or progress of anesthesia.

10 Flagg, loc. cit.

11. Guedel, loc. cit.

13. Lundy, J. S.: *Clinical Anesthesia* (Philadelphia: W. B. Saunders Co., 1942).

14. *Fundamentals of Anesthesia, Subcommittee on Anesthesia of Division of Medical Sciences,* National Research Council, 2nd ed. (Chicago: American Medical Association Press, 1944).

A precisely similar reflex is the eyelid reflex or, as Wartenberg[9] prefers to call it, the orbicularis oculi reflex. Its elicitation and evaluation during anesthesia is as significant as the jaw reflex,[10] except that it is not so resistant to general anesthesia, being usually depressed early in the third stage.[14] In the eyelid reflex, stretching of some portion of the orbicularis oculi muscle is the adequate stimulus, and contraction of the eyelids, the response (see Wartenberg's[9] account of numerous methods of applying the stimulus).

Another reflex which could well be placed in this category is Porter's reflex, as described by Flagg.[10]

All the preceding reflexes would tell the anesthetist no more than would serial study of the knee jerk, the biceps jerk, or indeed any other stretch reflex during the course of anesthesia. However, because the anesthetist is usually at the head of the table, the preceding stretch reflexes are more accessible to examination and hence more useful.

B. Skin-striated muscle reflexes

One of the previously mentioned reflex responses, the orbi-

9. Wartenberg, loc. cit.

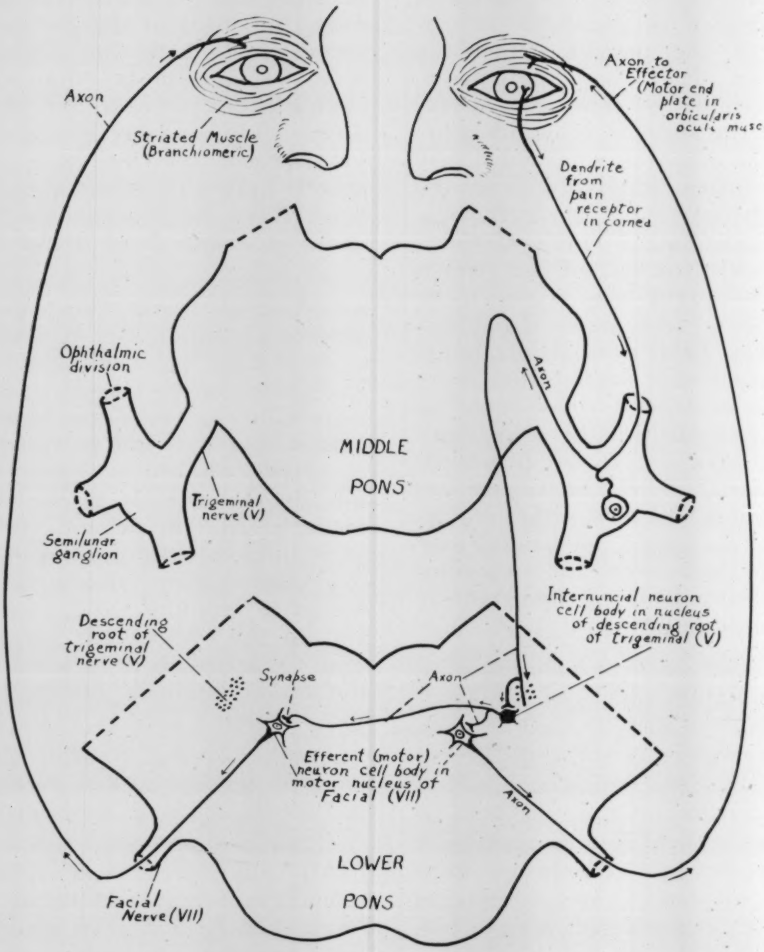


Fig. 4.—The orbicularis oculi reflex arc. Refer to text for further explanation.

cularis oculi reflex, consists of a contraction of the orbicularis oculi muscle in response to a stretch stimulus. The next reflex to be considered involves the same response, but the stimulus is exteroceptive in type—applied somewhere on the body surface. This is the corneal reflex. Here a painful stimulus applied to the cornea initiates the affair. The underlying reflex arc is represented in figure 4.[2,3] In this reflex it is to be noted that there is a bilateral response to a unilateral stimulus. This probably means that some of the activated internuncial neurons have collaterals which cross to the opposite motor

2. Krieg, loc. cit.
3. Fulton, loc. cit.

nucleus of VII, either directly (as represented in figure 4) or through the intermediation of other internuncials.

A precisely similar response will occur following tactile or painful stimuli to any part of the conjunctiva or even to the skin near the orbit, excepting that these responses will be depressed earlier by general anesthetics than will the corneal reflex.[7,10,11,12,13]

The foregoing reflexes are all excellent examples of a very restricted reflex discharge, protective in type, and may be collectively designated as skin-striated muscle type reflexes. They have their counterpart in the flexor withdrawal reflexes which can be elicited by appropriate skin stimulation over most of the body surface.[2,3,4,5]

The corneal reflex will usually be one of the last of the skin-striated muscle reflexes to fail with deepening anesthesia. This may be partly because of the important protective function it serves and also partly because all the central nervous system channels involved (fig. 4) are confined to the pons and medulla, the parts of the brain last depressed by general anesthetics. It should also return early in recovery.

Flagg[10] stated that a dangerous level of anesthesia and an active corneal reflex cannot coexist, and further that the corneal reflex is "the most valuable eye sign which we have." He also discussed the technics of applying the stimulus. Other anesthesiologists are somewhat more reserved (see Guedel,[11] Beecher,[12] Lundy[13]). Etsten and Himwich,[8] using pentothal, reported that the corneal reflex was not invariably absent until the third plane, but that it might disappear in the second stage or in the first or second plane.

In general, the corneal reflex reflects reasonably accurately the state of most of the other skin-striated muscle reflexes throughout the body. Consequently, it is of use to the anesthetist in gaging the depth and course of general anesthesia.

The skin-striated muscle type reflex can be used to judge accurately the effectiveness and extent of regional and spinal anesthesia in a patient whose conscious centers are somewhat numbed by preoperative sedation. Here, of course, the stimulus (e.g., pinching the skin with a hemostat) would be applied in and around the region being anesthetized, and the muscle response, or lack of it, observed. The surgeon's occasional mechanical stimulation of a mixed nerve, with consequent muscle contraction, should not be confused with an active skin-striated muscle reflex.

C. Pupillary reflexes

Two reflexes involving pupillary activity are of value to the anesthetist. One is the light reflex, in which the pupil contracts

7. Goodman, loc. cit.
10. Flagg, loc. cit.
11. Guedel, loc. cit.
12. Beecher, loc. cit.
13. Lundy, loc. cit.
2. Krieg, loc. cit.
3. Fulton, loc. cit.
4. Howell, loc. cit.
5. Macleod, loc. cit.

8. Etsten, loc. cit.

when a bright light is flashed into the eye. The other is the so-called "ciliospinal" reflex, in which a brief dilation of the pupil is the response to a painful (sometimes a tactile) stimulus.[3,6,10,11,12] These two reflexes are intimately interrelated, and both their reflex arcs are adequately shown in figure 5.

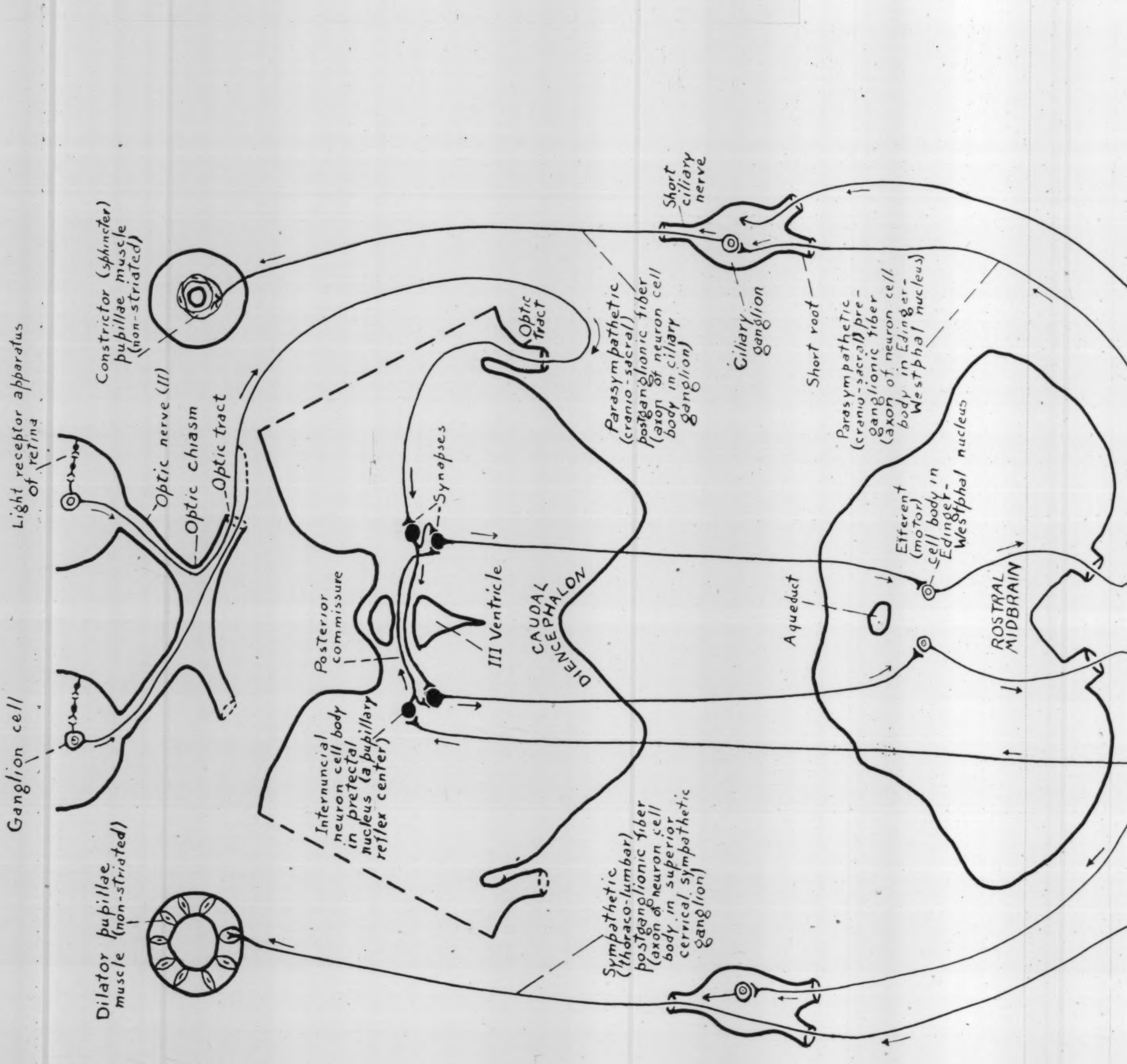
The upper right portion of figure 5 illustrates the light reflex arc. Although the response here is relatively restricted, the reflex arc cannot be diagramed adequately without calling into play greater numbers of neurons than we have had to use previously. Furthermore, the response is carried out entirely by smooth muscle cells, and the stimulus is picked up by an organ of special sense.

The ciliospinal reflex is adequately represented on the lower half and upper left side of figure 5. The initiating painful stimulus may be applied anywhere in the body, to visceral as well as to somatic receptors.[8,13,14] Since it is usually more convenient for the anesthetist to apply a stimulus to head or neck, the diagram considers that he elicits the response by pinching the skin of the neck below the ear (see fig. 5). The response here, as in the light reflex, is bilateral.

Another method of getting the same response to the same painful stimulus is illustrated in figure 5. Here the axon of the other internuncial cell, shown receiving the incoming nerve impulse initiated at the pain receptor, crosses the midline and turns cephalad in the spinotectal tract (see fig. 5). It runs as a part of this tract to the cephalic portion of the midbrain, where it synapses with pretectal area neurons, exerting an inhibitory effect on them. If they are inhibited, the Edinger-Westphal nucleus stops discharging to the constrictor muscle of the pupil in response to the light entering the retina (see fig. 5). This allows the constrictor muscle of the pupil to relax and the pupil to dilate. Regardless of which pathway is taken to secure it, the dilation is usually of brief duration. Obviously, if the pupils dilated, more light would strike the retina and set up an active light reflex, which would speedily tend to return the pupil to the status quo, unless the painful stimulus were very strong and continuously applied. Of course, if the centers involved were in a central inhibitory state, as is usually true in the lower planes of third stage anesthesia, the pupil would tend to be dilated and fixed, regardless of any stimuli presented.

This dual control of the size of the pupil would demonstrate what is commonly called the "antagonistic" action of the sympathetic and parasympathetic portions of the nervous system. Actually, they work closely together to maintain the pupillary size optimal under varying conditions. In order to do this most efficiently, the respective dilator and constrictor centers

3. Fulton, loc. cit.
6. Best, loc. cit.
10. Flagg, loc. cit.
11. Guedel, loc. cit.
12. Beecher, loc. cit.
8. Etsten, loc. cit.
13. Lundy, loc. cit.
14. Loc. cit.


Ganglion cell
Light receptor apparatus of retina
Optic nerve (II)
Optic chiasm
Optic tract
Constrictor (sphincter) pupillae muscle (non-striated)
Optic tract
Parasympathetic (cranio-sacral) postganglionic fiber (axon of neuron cell body in ciliary ganglion)
Short ciliary nerve
Ciliary ganglion
Short root
Parasympathetic (cranio-sacral) pre-ganglionic fiber (axon of neuron cell body in Edinger-Westphal nucleus)
Westphal nucleus
Synapses
Posterior commissure
III Ventricle
CAUDAL DIENCEPHALON
Efferent (motor) cell body in Edinger-Westphal nucleus
Aqueduct
ROSTRAL MIDBRAIN
Internuncial neuron cell body in pretectal nucleus (a pupillary reflex center)
Dilator pupillae muscle (non-striated)
Sympathetic (thoraco-lumbar) postganglionic fiber (axon of neuron cell body in superior cervical sympathetic ganglion)

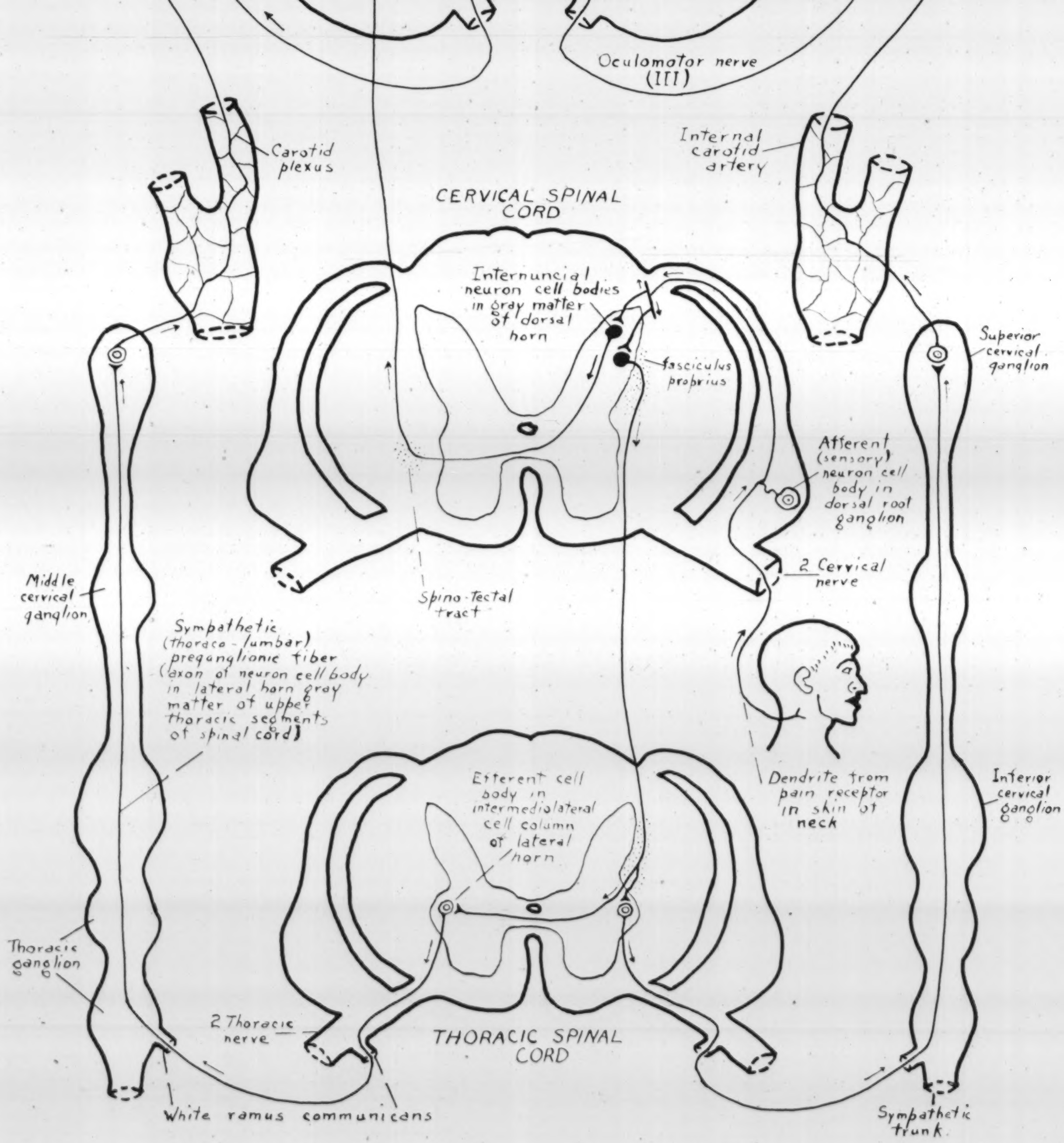


Fig. 5.—The pupillary reflex arcs. Refer to text for further explanation.

should be interrelated centrally, so that each time a burst of dilator impulses passes out the sympathetic division, inhibitory impulses should pass to the pretectal area and throttle it down, and vice versa. There is evidence that this sort of central interrelationship exists (see Gellhorn[15]). Thus a specific response, in this case pupillary dilation, can be brought about by increased discharge of the neurons controlling the dilator muscle or by inhibition of the neurons controlling the constrictor muscle. This is probably true of other reflexes as well.

Dilation of the pupil associated with strong emotions such as fright is well known. By some, this is attributed to hypothalamic and/or cortical activity impressing itself on the visceral efferent system. This could well explain the dilation frequently observed during induction of anesthesia.

If the anesthetist can elicit pupillary dilation in response to painful stimuli (and sometimes even if he cannot), the same or similar responses can be initiated, the depth of anesthesia permitting, by the maneuvers of the surgeon or his assistants. By anticipating such maneuvers, and increasing the depth of anesthesia accordingly, the patient can be better controlled. When responses are initiated in this way, but are unanticipated, the anesthetist should be careful not to misread them as evidences of rapidly lightening anesthesia. This is especially to be watched when the surgeon is working in the upper thorax, the upper abdomen, or the pelvis.

The fact that the pupil usually remains constricted after the preoperative use of morphine must be considered in interpreting pupillary reflexes. This opens the field of the effects of drugs on reflexes. Space permits but brief allusion to a few salient points. There is evidence that the passage of the nerve impulse across the synapse is associated with the production of acetylcholine at that point.[3,4,16] Acetylcholine is apparently also released at all somatic and parasympathetic (and at certain sympathetic) effector endings when a nerve impulse reaches them. On the contrary, at most sympathetic effector endings a substance closely akin to adrenalin is produced on receipt of a nerve impulse. Acetylcholine is a short-lived substance, being rapidly inactivated by cholinesterase at the site of production, hence making for localized, briefly acting responses. The adrenalin-like substances are long lived and therefore can circulate in the blood stream and cause widespread and sustained responses of the sympathetic system. Today, a host of drugs are available which depress, which enhance, or which mimic the action of the sympathetic or parasympathetic portions of the nervous system in a variety of ways. Certain of these the anesthetist must know about and use intelligently when indicated. Since routine preoper-

15. Gellhorn, E.: *Autonomic Regulations* (New York: Interscience Publishers, Inc., 1943).

3. Fulton, loc. cit.
4. Howell, loc. cit.
16. Nachmansohn, D: Chemical mechanism of nerve activity. Annals of the New York Academy of Sciences 47: 375-602, Dec., 1946.

ative medication makes use of certain of them (e.g., scopolamine, atropine), the anesthetist must keep in mind what drugs have been given and anticipate how they will affect the reflexes on which he routinely depends. It has recently been stressed that many of the anesthetic agents, themselves, have druglike actions on sympathetic or parasympathetic systems.[14]

D. The laryngeal reflex

The laryngeal reflex arc is shown in the portion of figure 7 set off by heavy broken lines. Typically, it is a reflex involving only centers located in the medulla. Likewise, it is a most important protective reflex for preventing the aspiration of foreign material into the tracheobronchial tree. The more usual stimuli may be the irritant action of the anesthetic gas on the mucous membrane or pus, blood, mucus, or vomitus in the pharynx and in danger of being aspirated. This reflex is not usually completely depressed until dangerous levels of anesthesia have been reached.[12,13,14] The laryngeal reflex is most active during induction and may interfere sufficiently with the anesthetic's reaching the lungs to delay satisfactory surgical anesthesia and to make the induction stormy. Its presence in the lower planes of anesthesia suggests that blood, or mucus, etc., is collecting in the airway and should be removed. In these lower planes of anesthesia the laryngeal reflex may also be elicited by surgical manipulations in neck, thorax, and abdomen which strongly stimulate interoceptors related chiefly to the vagus nerve.

12. Beecher, loc. cit.
13. Lundy, loc. cit.
14. Loc. cit.

E. The salivation reflex

The reflex arc concerned here is illustrated in the upper left portion of figure 6, as a part of the more widespread vomiting reflex arc. The salivation reflex is most likely to be troublesome during induction, the stimulus being the irritant effect of the anesthetic gas on the mucous membranes of nose, mouth, pharynx, and larynx. One reason for giving atropine preoperatively is to decrease the secretion of these glands.

In general, these more restricted reflex responses indicate to the anesthetist two things: (1) the general state of that *type* of reflex over the body as a whole, and (2) the physiologic state or condition of those portions of the central nervous system where the necessary centers are located. They are more useful after induction, as guides to the progress of anesthesia through the planes of the third stage and in the maintenance of anesthesia at the desired plane. Likewise, they chart the progress of recovery.

II. Reflexes involving more widespread and generalized discharges of both somatic and visceral effectors

Anesthesiologically pertinent examples of this type of reflex are coughing, hiccoughing, vomiting, sneezing, and swallowing. Since all of these make common use of many of the same muscles (final common path), since their co-ordinating mechanisms in the central nervous system are very similar, and since many of the

stimuli calling them forth are alike, we will consider only the vomiting reflex as an example of this group.

The vomiting reflex arc is represented in figure 6. It assumes a stimulus applied to one side of the nasopharynx, such as a nasal catheter introduced when the reflex could be elicited. This would activate an interoceptor in the nasopharynx, and the resulting nerve impulse would pass by way of the glossopharyngeal nerve to the vomiting center located in the medulla.* In response to the stimulus there will be a sudden inspiration (lower right of fig. 6), then closure of the glottis (middle right of fig. 6), followed by forced expiration (lower right of fig. 6), reversed peristalsis (lower left of fig. 6), sudden opening of the glottis (middle right of fig. 6—inhibition of final common path neurons to adductor muscles), and marked salivation (upper left of fig. 6).

It is obvious that the vomiting reflex requires the co-operative action of centers located well down in the spinal cord, as well as of those in the pons and medulla. From that fact and the general effect of anesthetics on the nervous system (see page 108), it follows that vomiting could only occur relatively early in anesthesia. Such is the case, this reflex being most troublesome near the end of the second stage. The same would hold true for the other reflexes of this type. As a rule, the anesthetist wishes the patient to pass rapidly through the stage when these reflexes are readily elicited. However, in some types of surgery, especially in the chest and about the neck, it is desired not to depress the cough reflex too much, if at all, so as to benefit from its protective guarding of the airway from aspiration.

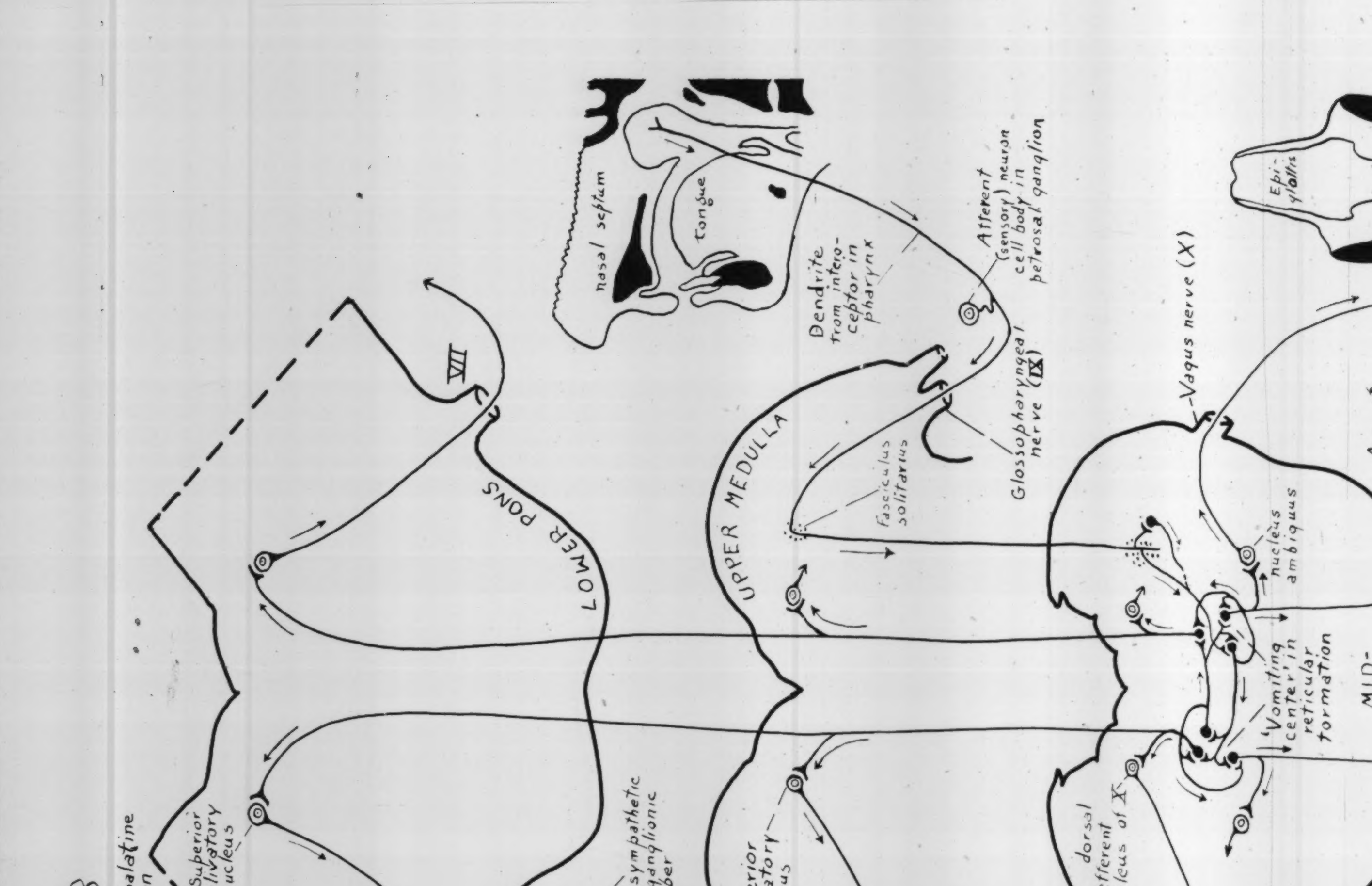
The vomiting reflex nearly always appears during recovery from, if not during induction with, general anesthetics. Flagg[10] noted that in some patients, during recovery, the vomiting reflex may be initiated by odors, for example, the odor of the anesthetic agent itself. He used pleasant smelling aromatic oils on strips of gauze placed below the patient's nose in such cases and found them helpful.

III. Reflexes characterized by rhythmic, continuous activity

These comprise the respiratory and circulatory reflexes. Detailed consideration of either of these reflexes or of the interrelationship between them would require a large volume and will obviously be impossible here. Figure 7 is an attempt to illustrate the respiratory reflex arc. Use is again made of the concept of centers (see footnote, this page), one in the pons and two in the medulla, all three being interconnected. The inspiratory center in the medulla may be thought of as tending to be continuously

*The term "center" as used in connection with these more complicated reflexes refers to a group or groups of what might be thought of as key internuncials which co-ordinate the parts of the total reflex with each other. As such, they must not be thought of as occupying a too circumscribed location, but rather as being more or less separated from each other. In some, and the vomiting center is one, drugs can directly stimulate the center.

10. Flagg, loc. cit.


nasal septum
tongue
Dendrite from intero-ceptor in pharynx
Afferent (sensory) neuron cell body in petrosal ganglion
Glossopharyngeal nerve (IX)
Vagus nerve (X)
Epi-glottis
VII
LOWER PONS
UPPER MEDULLA
fasciculus solitarius
nucleus ambiguus
Vomiting center in reticular formation
MID-
dorsal efferent nucleus of X
Superior salivatory nucleus
Inferior salivatory nucleus
Lacrimal
Sphenopalatine ganglion
Parasympathetic postganglionic fiber
Glands of nasal mucosa
Parasympathetic preganglionic fiber
Postganglionic parasympathetic fiber
Submax.
Submaxillary ganglion
Otic gang.
Parotid gland
Sympathetic postganglionic fiber
Carotid plexus
Parasympathetic preganglionic fiber
Sympathetic preganglionic fiber from upper thoracic segments of spinal cord
Sympathetic trunk

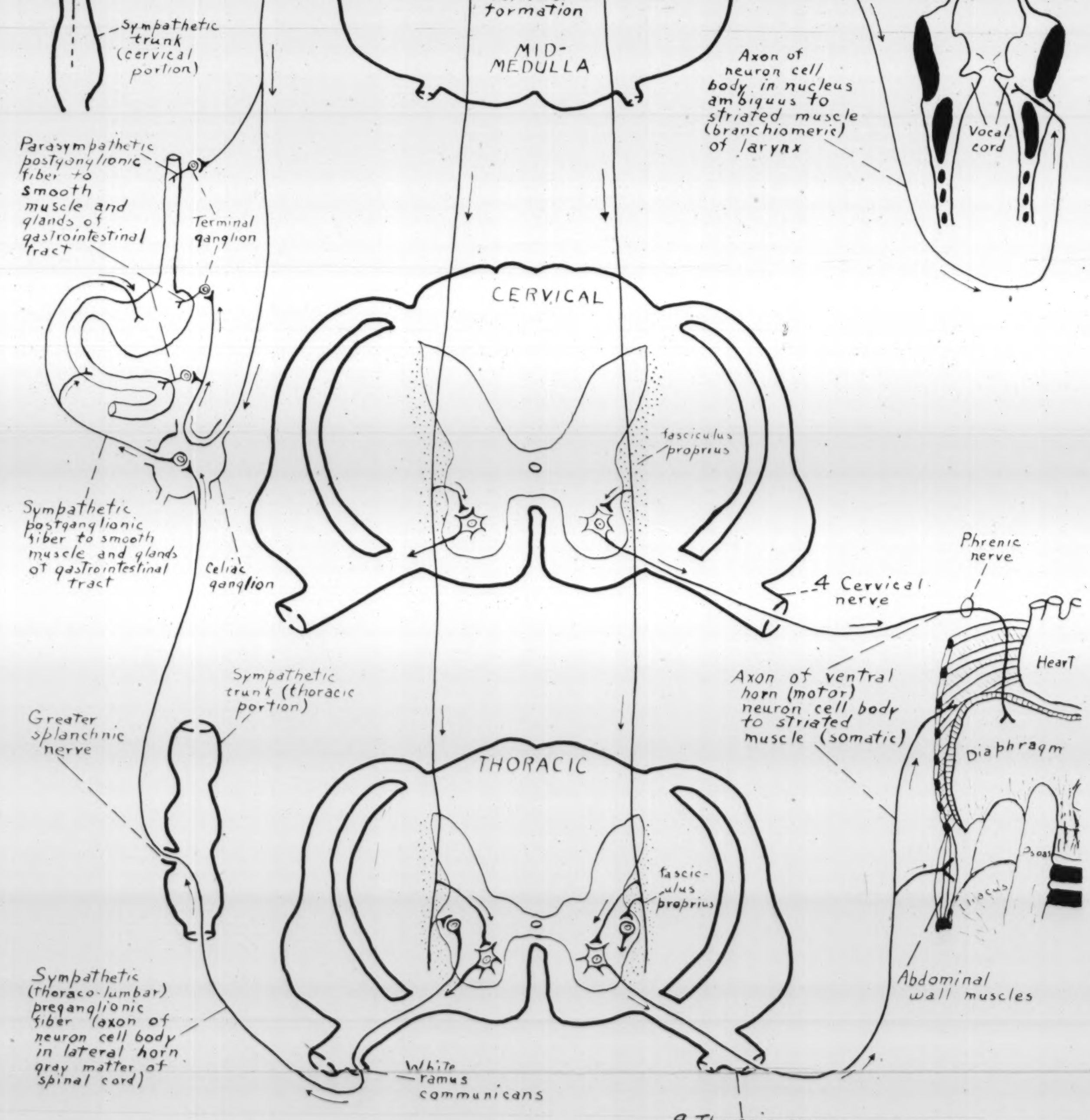


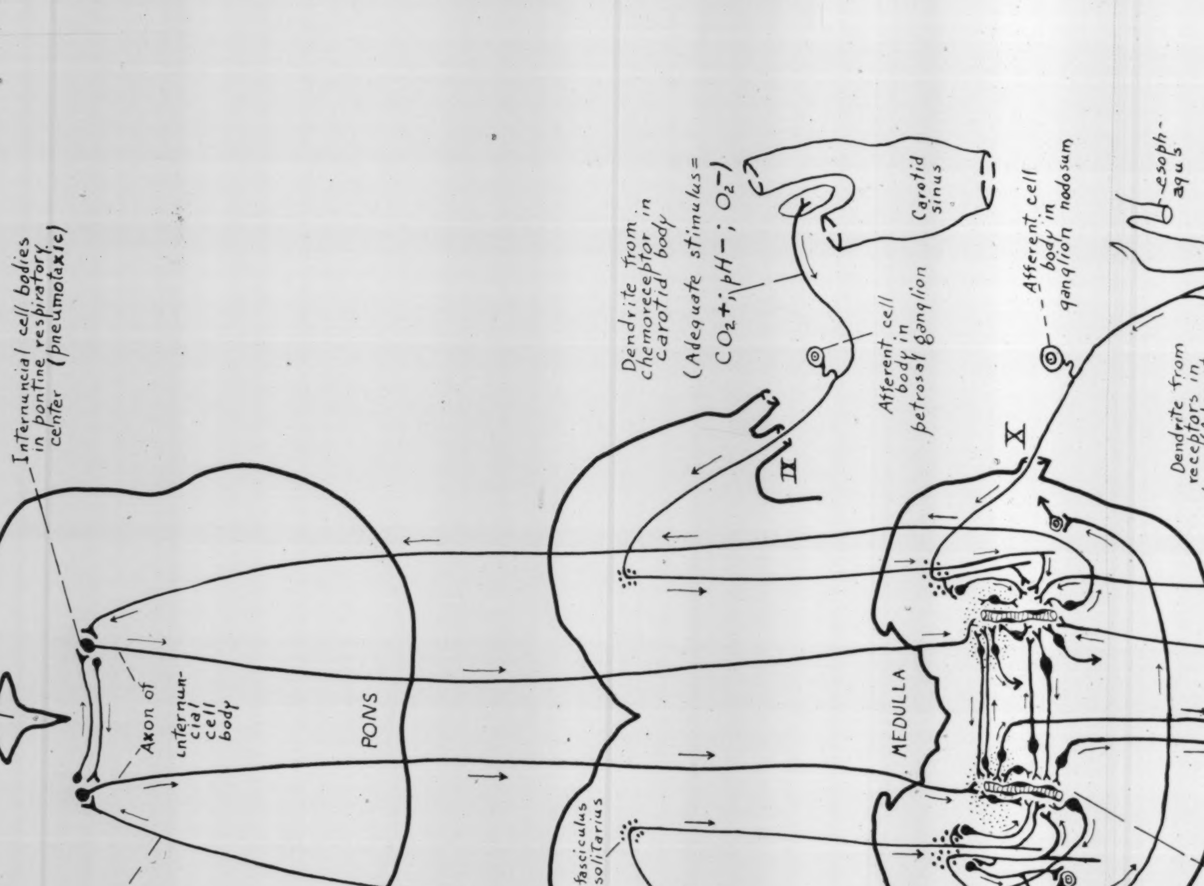
Fig 6.—The vomiting reflex arc. Refer to text for further explanation.

active and discharging the final common path neurons to the diaphragm and other inspiratory muscles (fig. 7, lower left). As the lung stretches during inspiration, stimuli stream up the vagus (fig. 7, middle right) and eventually inhibit the activity of the inspiratory center (fig. 7, middle), so that passive expiration follows automatically. The pneumotaxic center in the pons acts in a fashion analogous to the vagal afferents (fig. 7, upper part), that is, it inhibits the inspiratory center and thus provides for rhythmic respiration when the vagal afferents are not operating efficiently. Since, under these circumstances, the pneumotaxic center is discharged by impulses sent out by the inspiratory center, itself, but in turn inhibits the inspiratory center, we have here an example of what physiologists term a "reverberating circuit" within the central nervous system (fig. 7, upper part). Such circuits appear to be adequately organized to insure continuous rhythmic activity.

As reference to figure 7 will show, there are many other impulses normally streaming into the respiratory center in response to a variety of stimuli throughout the body. These can and do modify the activity of that center. Probably the most important of these, from the standpoint of regulating respiratory activity to best serve body needs, are those initiated in the chemoreceptors, the carotid and aortic bodies (fig. 7). Increased concentrations of carbon dioxide, increased acidity, and decreased concentrations of oxygen in the blood circulating through these chemoreceptors serve as adequate stimuli to initiate nerve impulses which discharge to the respiratory center (fig. 7). They tend to modify the activity of that center so as to correct the situation by appropriate changes in the rate, the depth, and the rhythm of respiration. In addition, the center itself is sensitive to the concentration of the carbon dioxide in the blood which perfuses it.

Painful and proprioceptive stimuli are also very important in modifying the activity of the respiratory center. These, of course, would not be very active in lower planes of surgical anesthesia. In such planes, then, it is obviously the chemoreceptors, the vagal afferents, and the inherent activity of the center itself which must be operating the respiratory mechanism.

During induction there is likely to be much variation in the respiratory picture, and here the anesthetist is most interested in maintaining an open airway so that a sufficient volume of the gaseous anesthetic agent will reach the lungs to induce surgical anesthesia. Quite obviously, as suggested by the foregoing information and that available in figure 7, during surgical anesthesia respiration would be likely to be regular and deep, excepting when surgical maneuvers were carried out in the neck, the thorax, and the upper abdomen or where there was interference with the airway. Intercostal muscle paralysis, appearing in the third plane, is one of the most definite changes in respiratory reflex activity here. It speaks for decreased respiratory volume and


Internuncial cell bodies in pontine respiratory center (pneumotaxic)
IV Ventricle
Axon of internuncial cell body
PONS
Axon of internuncial cell body in inspiratory center
fasciculus solitarius
MEDULLA
Dendrite from chemoreceptor in carotid body
(Adequate stimulus= CO_2+; pH−; O_2−)
Carotid sinus
Afferent cell body in petrosal ganglion
IX
X
Afferent cell body in ganglion nodosum
esophagus
Carotid body
Dendrite from presso-receptor in carotid sinus
(Adequate stimulus= change in blood pressure)
Dendrite from laryngeal mucosa
Axon
LARYNGEAL REFLEX

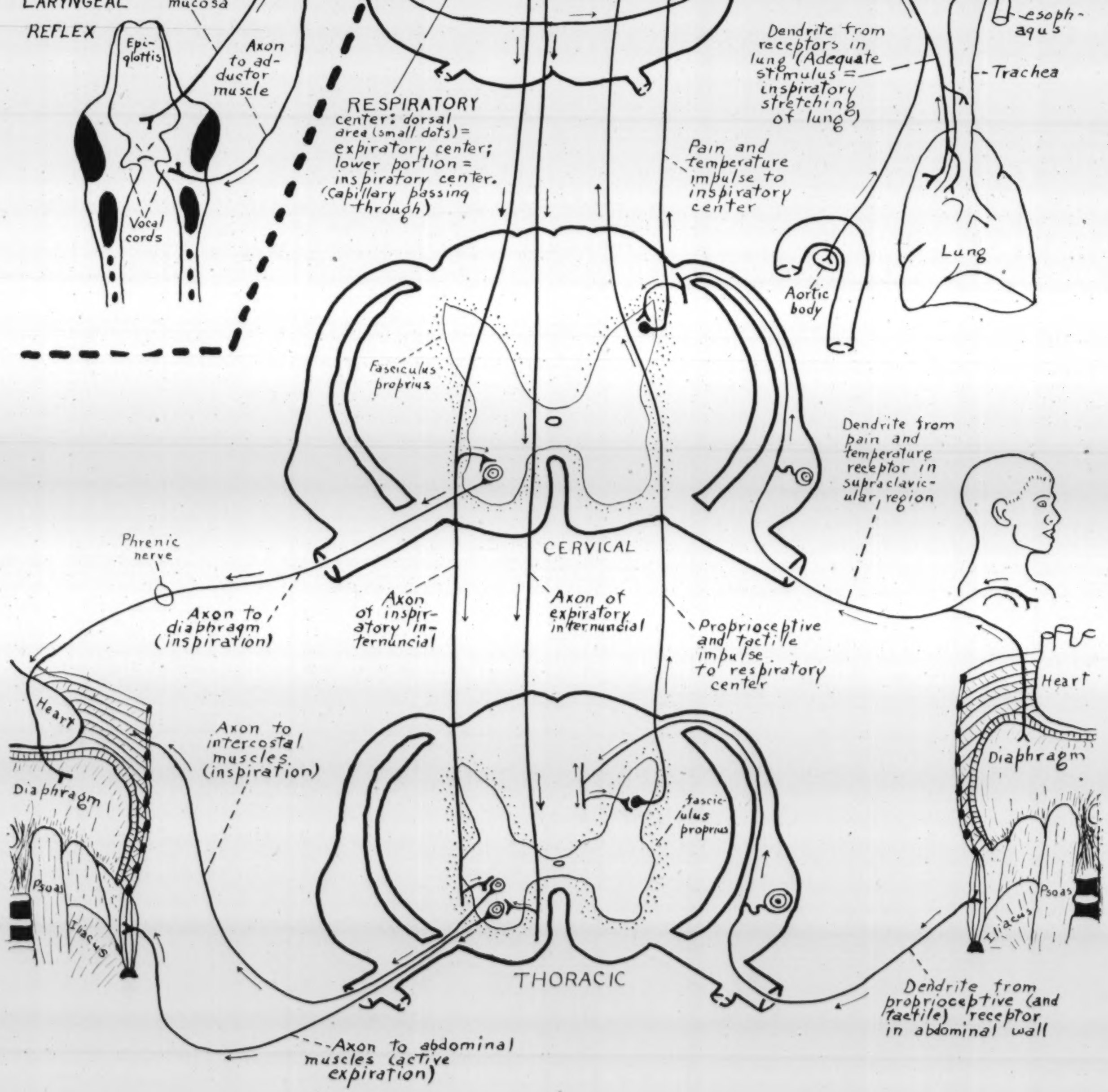


Fig. 7.—The respiratory reflex arc. Refer to text for further explanation.

beginning fourth plane anesthesia. During the fourth plane, minute volume further decreases, with attendant complications of hypoxia, and as the lower limits of this plane are reached, respiratory activity ceases altogether.[10,11,12,13,14] In these lower planes of surgical anesthesia the respiratory (and circulatory) reflexes are the only guides the anesthetist has. During intrathoracic operations they must be watched with particular care and evaluated accurately, for here there is certain to be much opportunity for strong stimulation of the vagal interoceptors. The impulses streaming in from these may well rapidly depress the efficiency of the respiratory and circulatory centers to dangerous levels.

A diagram similar to figure 7 could be constructed to illustrate many of the influences operating on the circulatory center. It would make use of many of the same nuclei as the foregoing. The heart and blood vessels and their controlling nervous system mechanisms would be major additions to the picture. What has been said relative to the respiratory reflexes would apply equally well, in general, to the circulatory reflexes in anesthesia. The activity of the circulatory center will modify the rate, force, and rhythm of the heart and the tone of the vascular walls. This will be expressed to the anesthetist chiefly through the pulse, the blood pressure, and the color of the patient. Whereas the chemoreceptors (carotid and aortic bodies, fig. 7) are most important aids in regulating the respiratory center, the pressoreceptors (e.g., carotid sinus, fig. 7) are most important in regulating the circulatory center.

SUMMARY

1. The more important anatomic and physiologic characteristics of reflexes have been briefly considered.

2. The various general types of reflexes of value in anesthesiology have been described, and the reflex arcs for certain of them have been illustrated in detail.

3. The relationships of these reflexes to anesthesia and their importance to the anesthetist have been briefly discussed.

10. Flagg, loc. cit.
11. Guedel, loc. cit.
12. Beecher, loc. cit.
13. Lundy, loc. cit.
14. Loc. cit.

A REVIEW OF INHALATION THERAPY

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Until recently, the term inhalation therapy was restricted, for the most part, to the administration of oxygen. Because the medical profession in general was unaware of its advantages and unfamiliar with technics of administration, it became popular only as a heroic measure to be used in the moribund patient. During the past decade, rapid strides have been made in new technics and above all in educating the doctor and the nurse. The advent of chemotherapy and antibiotics has opened up new fields in the treatment of respiratory tract diseases. It is the purpose of this article to discuss the rationale and technics of this field of therapy. In the discussion, I will touch upon:

I. Oxygen administration
II. Oxygen helium mixtures
III. Positive pressure breathing
IV. Aerosol therapy

Inhalation therapy is based upon a fundamental knowledge of the physiology of respiration. Ordinarily, breathing is an effortless and unconscious process. Consequently, no illness is more alarming to the patient than one which provokes difficulty in breathing.[1]

To the ancients, air was one of the four elements of which the earth was composed. It was Joseph Priestly in 1775 who discovered oxygen by heating red oxide of mercury. He remarked on his breathing a large jar of oxygen, "I fancied that my breath felt peculiarly light and easy for some time afterwards. Who can tell but that in time this pure air may become a fashionable article in luxury." Shortly thereafter, the French scientist Lavoisier laid down the fundamental principles of breathing by demonstrating that oxygen is absorbed through the lungs, and that carbon dioxide and water vapor are given off in expiration. Within 25 years, the discovery of oxygen was put to use clinically by Dr. Thomas Beddoes, of Bristol, who gave inhalations of oxygen to patients with asthma and heart disease. He made breathing bags of oiled silk, but the technic of administration was inadequate.

Until the latter half of the nineteenth century, there was no attempt at correlating disease

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1. Barach, A. L.: *Principles and Practices of Inhalation Therapy* (J. B. Lippincott Co.: Philadelphia, 1944).

with lack of oxygen. But following a series of balloon ascensions in 1862, several untoward symptoms developed above altitudes of 15,000 feet. This was confirmed by experimentation in decompression chambers, mountain climbing expeditions, and through inhalation of low oxygen mixtures.[2,3,4] It became apparent that many of the symptoms in disease states were identical with those produced in normal subjects on low oxygen intake. In pneumonia, heart failure, asphyxia, and a variety of other conditions, it was found that the patients suffered from a serious degree of oxygen want, or anoxia. It was demonstrated in such patients that the pressure of oxygen in the arterial blood was greatly diminished, and that many of the symptoms would disappear when the patient inhaled 40-60 per cent oxygen, which raised the oxygen saturation of blood to normal or near normal values.[5,6,7]

The development of efficient methods of administering oxygen was required for improving inhalation technic. In the interval between the two World Wars, the following methods were developed: (a) catheter, (b) mask, (c) oxygen chamber or room, and (d) tent. In general it has been found that 50 per cent oxygen is usually required to raise the oxygen concentration in the arterial blood to a level near that of normal. However, higher concentrations are of definite advantage to certain patients suffering from severe anoxia.

It is not my intention to go into a detailed account of the various pathologic processes in which oxygen therapy would be helpful, but a discussion of the value, indications, and choice of procedures is necessary for a better understanding and employment of inhalation technics.

Acute Anoxia

The simplest form of acute oxygen want is found in altitude sickness, mines and mining accidents, and anesthesia employing low oxygen mixtures.[8] The brain is the organ most sensitive to lack of oxygen, and symptoms of dulness and irritability are soon followed by headache, drowsiness, loss of vision, paralysis, and unconsciousness. Human beings may die when exposed to complete asphyxia for 10 minutes. The heart and adrenal glands are also quite sensitive to oxygen lack.[9]

The treatment of acute anoxia is simple replacement. This is especially important in civil and military aviation where oxygen should be employed at altitudes over 10,000 feet. One hundred per cent oxygen will protect individuals up to 38,000 feet; beyond

2. Bert, P.: *La Pression Barometrique, Recherches de Physiologie Experimentale* (Paris: Masson et Cie, 1878).
3. McFarland, R. A.: The effects of oxygen deprivation (high altitude) on the human organism. Technical Div. Report No. 11; May 1938, Civil Aeronautics Authority, Washington.
4. Barach, A. L.: "Pilot error" and oxygen want with a description of a new oxygen fore tent. J.A.M.A. **108**:1868, 1937.
5. Stadie, W. C.: Treatment of anoxemia in pneumonia in an oxygen chamber. J. Exper. Med. **35**:337, 1922.
6. Barach, A. L., and Woodwell, M. N.: Studies in oxygen therapy with determination of the blood gases. Arch. Int. Med. **28**:367, 1921.
7. Meakins, J. C.: Oxygen-want: Its causes, signs and treatment. Edinburgh M. J. **29**:142, 1922.
8. Barach, A. L., and Rovenstine, E. A.: The hazard of anoxia during nitrous oxide anesthesia. Anesthesiology **6**:498, 1945.
9. Van Liere, E. J.: *Anoxia, Its Effect on the Body* (Chicago: University of Chicago Press, 1942).

this altitude, pressurized cabins become necessary.[10]

PNEUMONIA

Most of you have had occasion to see oxygen used in the treatment of pneumonia. In fact, prior to the use of antiserums and chemotherapeutic and antibiotic agents, such as the sulfonamides and penicillin, oxygen was the mainstay of such therapy. As a result of pulmonary inflammation, ventilation in the lungs is inadequate, and the blood passes through air cells which contain little or no oxygen. Increased pulse rate is the outstanding sign of lack of oxygen, but other symptoms, such as cyanosis, delirium, dyspnea, and insomnia, are equally important. Through the inhalation of oxygen, anoxia is overcome, and the aforementioned symptoms are relieved. In numerous studies the inhalation of oxygen was shown to be responsible for prolonging the lives of seriously ill patients.[5,6,7,11,12]

The percentage oxygen to be used in the treatment of pneumonia must be individualized, but in general between 40 and 60 per cent seems adequate for the majority of patients. The selection of equipment to be used depends on the training of the personnel administering the oxygen, and on the patient himself.[1]

10. Armstrong, H. G.: *Principles and Practice of Aviation Medicine* (Baltimore: Williams and Wilkins Co., 1940).
5. Stadie, loc. cit.
6. Barach, loc. cit.
7. Meakins, loc. cit.
11. Bullowa, J. G. M.: *The Management of the Pneumonias* (New York: Oxford University Press, 1937).
12. Barach, A. L.: Oxygen therapy in pneumonia. New York State J. Med. **29**:985, 1929.
1. Barach, loc. cit.

1. A rubber catheter introduced into the nasopharynx is the simplest and least expensive equipment, and with a flow of 7 L. a minute, a concentration of 38 to 40 per cent oxygen may be obtained.

2. The most comfortable method for administering oxygen concentrations between 40 and 60 per cent is the oxygen tent. The newer vinolyte type allows ready inspection, and the feeling of claustrophobia which many patients experience is alleviated.

3. The oxygen room has certain advantages in the care of patients requiring prolonged oxygen therapy, but the expense has limited its usefulness.

4. A mask (oronasal or nasal type) is advantageous for administration of concentrations of oxygen between 70 and 100 per cent. This is especially useful in the treatment of severe distention, shock, hemorrhage, or cardiac failure.

The duration of oxygen treatment for pneumonia depends on the condition of the patient. It is usually safe to terminate therapy when the temperature has returned to normal and the pulse rate has decreased. If interruption of oxygen therapy brings on dyspnea and cyanosis, it must be resumed. In the presence of coexisting cardiac failure, the reduction in oxygen concentration should be gradual.

PULMONARY EDEMA

Pulmonary edema is one of the most common and serious causes of anoxia and may arise in the presence of heart disease, especially hypertension, pneumonia,

shock, gas poisoning (war and industrial), asthma, and obstructive lesions of the tracheobronchial tree.

The treatment of this condition depends on the underlying state, but insofar as inhalation therapy is concerned, the basis of treatment is to provide a sufficient amount of oxygen to sustain life, to diminish pulmonary congestion, and to oppose the tendency of serum to "leak out" of the capillaries by the application of positive pressure.[13]

The oxygen may be administered either by modified meter mask or by a positive pressure hood. Forty to 60 per cent oxygen is utilized with a positive pressure of 3-6 cm. of water. Customarily 4-6 cm. is employed until the edema is almost cleared and the pressure lowered at two to three hour intervals until atmospheric pressure is reached. It must be pointed out that the occurrence of pulmonary edema in peripheral shock is not associated with chronic passive congestion of the lungs, and that positive pressure should not be used and may even be disadvantageous. Administration of blood or plasma intravenously and 100 per cent oxygen is useful.[1]

Cardiac Disease

In cardiac failure, congestion of the lungs may take place owing to the inability of the heart to maintain the circulation. The circulation rate slows, and stagnation results in anoxia. In acute cardiac failure, the inhalation of 50-100 per cent is indicated, irrespective of the cause, and should be continued until the acute phase has passed. In chronic cardiac failure, oxygen has had a favorable effect in hypertensive and arteriosclerotic heart disease but little effect in rheumatic valvular disease.[14] Fifty to 70 per cent oxygen is used continuously for three to six weeks. This therapy is combined with other measures for treating cardiac failure, and when symptoms have subsided, the oxygen concentration is gradually lowered to 40 per cent and then 30 per cent over a period of five days. In the selection of the method, the comfort of the patient is of the utmost importance.

Acute coronary thrombosis results in closure of a coronary blood vessel with death of the heart muscle due to lack of oxygen. The inhalation of 50-100 per cent oxygen is urgently indicated. The relief of pain is often striking, difficulty in breathing and restlessness are diminished, and Cheyne-Stokes breathing, if present, is sometimes abolished.[15,16] In severe conditions, oxygen should be given for five or more days after the onset of the attack.

Bronchial Asthma, Emphysema, and Related Pulmonary Disturbances

Asthma is an allergic phenomenon resulting in the production

13. Barach, A. L.; Martin, J., and Eckman, M.: Positive pressure respiration and its application to the treatment of acute pulmonary edema. Ann. Int. Med. 12:754, 1938.
1. Barach, loc. cit.

14. Barach, A. L., and Richards, D. W., Jr.: Effects of oxygen therapy in congestive heart failure. Arch. Int. Med. 48:325, 1931.
15. Levy, R. L., and Barach, A. L.: The therapeutic use of oxygen in coronary thrombosis. J.A.M.A. 94:1363, 1930.
16. Boland, E. W.: Oxygen in high concentration for relief of pain in coronary thrombosis and severe angina pectoris. J.A.M.A. 216:203, 1937.

of bronchospasm and edema and causing an increased resistance to the flow of air in and out of the lung. The disease occurs in attacks manifested by a wheezing type of breathing and when severe and continuous is called status asthmaticus. Removal of the offending allergen may effect a cure, but treatment of the attack by inhalation therapy becomes necessary for symptomatic relief. While the inhalation of oxygen will increase the blood oxygen, it does not relieve the dyspnea, since this is due to the mechanical effort necessary to ventilate the lungs in the presence of obstruction. The application of the laws of physical chemistry has resulted in the use of helium-oxygen mixtures. It is known that the velocity of movement of a gas is in inverse proportion to its molecular weight; therefore, the lighter the gas, the easier it will be for it to pass a point of obstruction. A mixture of 20 per cent oxygen and 80 per cent helium is one third as heavy as air. In status asthmaticus, the ideal method is to employ a mixture of 30-35 per cent oxygen in helium under a positive pressure of 3-6 cm. of water, by either hood or mask.[1] This mixture is administered for two hours three times daily for three to five days. In approximately 70 per cent of a series, this treatment resulted in subjective relief of the dyspnea and made the patient more responsive to the administration of bronchodilators such as adrenalin and aminophylline.[17] If helium and oxygen are not available, 100 per cent oxygen under positive pressure is useful.

Patients with obstructive lesions of the upper respiratory passages, such as occur in tracheobronchitis, croup, and cancer of the bronchi, may be benefited by the described therapy.

Pulmonary emphysema is a progressive disease accompanied by loss of elasticity of the lung and dilatation of the air sacs. Accompanying this, there is usually some degree of bronchospasm. Treatment of this condition by the inhalation of 50 per cent oxygen is palliative at best in senile emphysema, and improvement is not maintained on cessation of therapy, but when used in chronic hypertrophic emphysema, a regulated course of oxygen treatment may be of considerable value. If infection supervenes, oxygen may be indicated. It may become necessary to use positive pressure, bronchodilators, and aerosol therapy.

Inhalation therapy is an important and sometimes life-saving measure in the treatment of a wide variety of conditions. An incomplete list would include: (a) accidental asphyxia from drowning, electric shock, (b) asphyxia of the newborn, (c) lung collapse and atelectasis, (d) pulmonary infarction, (e) hemorrhage and shock, and (f) paralysis of muscles of respiration as in poliomyelitis.

Before concluding this section, I should like to make two comments:

1. After extensive abdominal operations, the prompt institution of oxygen therapy tends to prevent the development of post-

1. Barach, loc. cit.
17. Barach, A. L.: Physiological methods in the diagnosis and treatment of asthma and emphysema. Ann. Int. Med. 12:454, 1938.

operative atelectasis and pneumonia. Ventilation of the lower portions of the lungs should be encouraged, and in order to permit free excursion of the diaphragm, no unnecessarily tight abdominal binders should be used.

2. A great deal has been written about the toxicity of 100 per cent oxygen. Experiments on lower animals seem to confirm this concept, but the human being appears to be more resistant to the toxic effect. However, in our present state of knowledge, caution should be exercised in the long continued use of pure oxygen: It is best to dilute it down to a 50 per cent mixture several times daily when using a hood or mask. For patients with pre-existing long continued anoxia and with cerebral arteriosclerosis, the immediate administration of 100 per cent oxygen may bring on a transient state of delirium, coma, and hyperexcitability.[1] The best method is to start with a dilute mixture and gradually raise the concentration over a period of hours.

Aerosol Therapy

In recent years, inhalation therapy has included the use of aerosols in addition to the more conventional therapeutic gases. The administration of drugs by inhalation is as ancient as the art of medicine itself, and the use of steam medicated with essential oils was a common practice among the ancient Greeks. With the advent of Lister's theory of disinfection, germicidal sprays and vapors became popular on the continent. However, the use of dilute solutions of carbolic acid was soon discarded, but with the introduction of newer knowledge concerning airborne or droplet infection, a search for more effective bactericidal mists resulted in the use of propylene glycol vapor and resorcinol.

It might be well to define certain terms in order to avoid confusion.

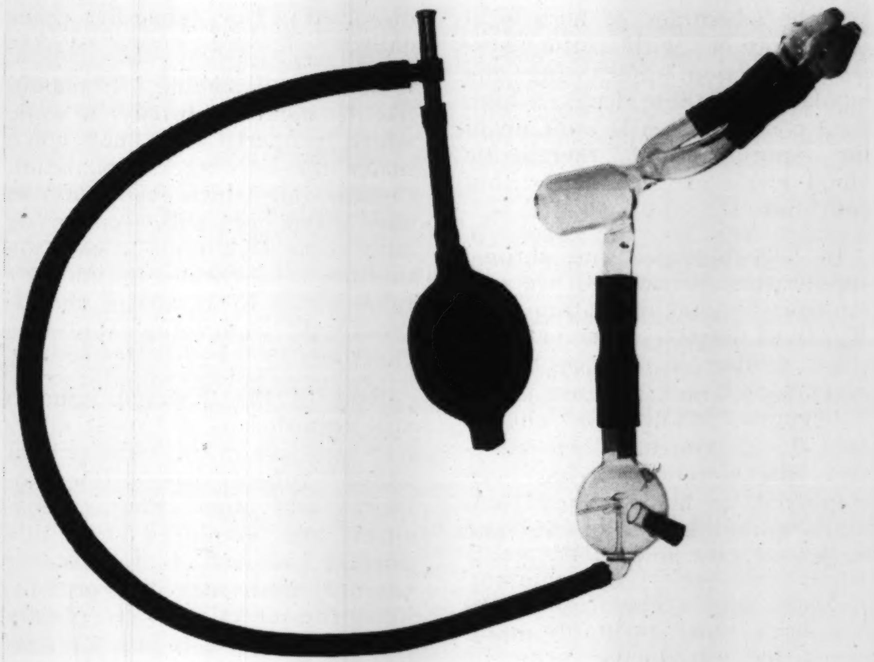
Aerosols are suspensions of either solid or liquid particles in a gas.

Atomization, in medical terminology, has come to mean the formation of a spray from a bulk liquid. Actually, an atomizer forms an aerosol of such large particle size that it is not stable and settles out rapidly.

Nebulization is the production of a stable aerosol of small particle size.

The rationale of aerosol therapy is the application of a suitable drug topically to diseased portions of the lung, bronchus, or sinus. To accomplish this, the particle or mist, in high enough concentration to be effective, must be deposited in the desired locale. In order to obtain such deposition, many factors must be considered, of which particle size is probably the most important. If particles of the mist are too large, they will be deposited on the pharynx, tongue, and trachea. If they are too small, they will be exhaled, and only 35 per cent will be deposited. Apparently, the ideal size ranges from 0.8 to 6 micron radius with mean average radius of 1 to 3 microns; such particles are deposited in the

1. Barach, loc. cit.



Apparatus for sinus aerosol therapy employing negative pressure with hand bulb.

lung from 63 to 96 per cent.[18,19,20,21] All appropriate nebulizers function on the principle evolved by Heubner of placing capillary tubes at right angles and using a stream of gas as the propellent.

The application of the aerosol technic to the treatment of disease received impetus when excellent results were obtained by using concentrations of epinephrine up to a 1:100 dilution. It was found that the inhalation of adrenalin mists was often as effective in asthma as epinephrine administered parenterally, with the advantage of relief from many unpleasant side reactions. In the past few months, we have been experimenting with certain synthetic drugs of the adrenaline family, such as N-iso-propyl adrenaline which Segal found of value in a considerable number of patients.[22]

With the advent of chemotherapeutic agents, such as the sulfonamides and, more recently, the antibiotics penicillin and streptomycin, a new field was opened to inhalation therapy. Bronchopulmonary infection and sinusitis could be approached by topical measures, since aerosols

18. Heubner, M.: Uber Inhalation zerstanfter Flussigkeiten. Ztschr. f.d. ges. exper. Med. 10:269, 1919.
19. Talbot, T. R.; Quimby, Edith H., and Barach, A. L.: A method of determining the site of retention of aerosols within the respiratory tract of man by the use of radioactive sodium. Am. J. M. Sc. 214:585, Dec., 1947.
20. Barach, A. L.; Bickerman, H. A., and Garthwaite, B.: Studies on aerosol deposition. Post-Grad. M. J., to be published.
21. Wilson, I.: Unpublished data.
22. Segal, M. S., and Bakey, J. F.: The use of isuprel for the management of bronchial asthma. Bull. New England Med. Center 9:62, 1947.

had the advantage of high local concentration with little systemic reaction. Following the inhalation of these drugs, a high local concentration is built up in the sputum, and therapeutic blood levels are attained within one hour.

In bronchiectasis and chronic suppurative bronchitis, we administer by oral inhalation from 50,000 to 100,000 units of penicillin diluted in 1-2 cc. saline every four hours, at flows of 6-7 L. oxygen, followed by one or two, 0.5 cc. rinses. When necessary, the meter mask has been employed, as has the head tent with more dilute solutions and larger oxygen flows.[20,22,23]

Acute and chronic sinusitis has been most favorably influenced by introducing penicillin aerosol by the intermittent negative pressure technic (see figure).[24] Before giving antibiotics, it is essential that sputum cultures and nasal smears be taken to determine the type of infecting organism and its sensitivity to the drug to be employed. Against the gram negative family, including Friedlander's bacillus, Hemophilus influenzae, and B. aerogenes, streptomycin has been more active. In the mixed infections common to bronchiectasis, a combination of penicillin and streptomycin has proved most successful. Olsen of the Mayo Clinic used 50,000 units of streptomycin and 20,000 of penicillin dissolved in 1 cc. saline five times daily.[25]

Before concluding, I should like to mention, briefly, a topic which properly does not come under the heading of inhalation therapy but which many of you may have, or will, sooner or later come in contact with: the treatment of chronic pulmonary tuberculosis by means of the alternating, equalizing pressure chamber.

Rest of the diseased lung is still regarded as the most effective principle in the treatment of active tuberculosis. Pneumothorax and other collapse measures are employed for this purpose, as well as for closing cavities that may be present. With the intention of completely immobilizing the lungs, a chamber was built which could apply a simultaneously equal force to the outer and inner surfaces of the chest wall by means of equalizing air pressures. This method provided adequate pulmonary ventilation without lung movement.[26]

The patient lies in a sealed chamber with a baffle plate between head and trunk. Positive and negative pressures of 1/6 of an atmosphere are applied 25 times per minute in order to obtain adequate ventilation. Thus far, patients have been selected who have been in advanced stages and for whom no other therapy was thought to offer opportunity for further benefit.

20. Barach, loc. cit.
22. Segal, loc. cit.
23. Garthwaite, B., and Barach, A. L.: Penicillin aerosol therapy in bronchiectasis, lung abscess and chronic bronchitis. Am. J. Med. **3**:261, 1947.
24. Barach, A. L., et al: Penicillin aerosol and negative pressure in the treatment of sinusitis. Am. J. Med. **1**:268, 1946.
25. Olsen, A. M.: Streptomycin in the treatment of chronic bronchiectasis. Proc. Staff Meet., Mayo Clin. **21**:53, 1946.
26. Barach, A. L.: Immobilization of both lungs produced by the equalizing pressure chamber with results of treatment in pulmonary tuberculosis. Ann. Int. Med. **26**:687, 1947.

Cessation of breathing is a voluntary act which the patient must learn during the early days of treatment. Approximately 30 per cent of patients cannot voluntarily control respiration and are unsuited for this trial. Approximately 12 hours per day for three to four months constitute a course of therapy. One of the striking effects is the degree of relaxation which takes place in the trained patient. For hours at a time, a state simulating hibernation is produced with no motion of any type. Improvement is manifested by a decrease in temperature and pulse, increase in weight, and a feeling of well being.

Although the series of patients so treated has been small, noteworthy success in arresting the process and closing the cavities has been attained in a significant number. As more alternating, equalizing pressure chambers become available for clinical studies, the effectiveness of this treatment for advanced pulmonary tuberculosis may be better evaluated.

SUMMARY

Inhalation therapy has evolved from early crude attempts to supply an oxygen-enriched atmosphere to the desperately ill patient into a well defined system of therapeutics based on a broader knowledge of respiratory physiology.

1. Oxygen administration to patients suffering from pneumonia, cardiac failure, shock, hemorrhage, and asphyxial states tends to decrease the anoxia. Technics employed depend on the concentration of oxygen desired, the comfort to the patient, and the training of personnel administering oxygen therapy.

2. In conditions characterized by bronchospasm and tracheobronchial obstruction, the use of lighter gas mixtures employing helium has a decided advantage over air in attaining adequate pulmonary ventilation.

3. The application of positive pressures by either mask or hood to the treatment of pulmonary edema generally results in prompt clearing of the rales and disappearance of edema both in acute cardiac failure (left ventricular insufficiency) and in pneumonia.

4. Aerosol therapy consists in the inhalation of a suitable drug into the sinuses and bronchopulmonary tree. The main indications are:

a. Production of bronchial dilatation in asthma and related conditions characterized by spasm.

b. Treatment of infection in respiratory disease, employing chiefly antibiotic agents, such as penicillin, and in some cases the sulfonamides.

5. Complete immobilization of the lungs with adequate pulmonary ventilation by means of an alternating, equalizing pressure chamber has been applied to the treatment of advanced pulmonary tuberculosis.

PRINCIPLES OF FLUID BALANCE FROM THE STANDPOINT OF THE ANESTHETIST

Robert Elman, M. D.*
St. Louis

My remarks will deal with fundamental considerations in regard to fluid balance in the body, because I know all of you are interested in the rationale of the things you do. Fluid administration during an operation should not be viewed simply as a mechanical procedure but as a means of dealing with some disturbance that has occurred or may occur in the patient's body. Often the disturbances are in water balance, and adequate treatment requires a fundamental knowledge of the behavior of water in the body.

In giving fluids during an operation, the intravenous route must be used, because many patients are given inhalation anesthetics and cannot take anything by mouth. Moreover, many patients require that the fluids be given rather rapidly, and the oral route cannot be trusted for rapid absorption. Another reason for using the intravenous route is that a needle is in place for an immediate transfusion if necessary. However, fluids should not be given for curative purposes only; fluids are not given just because the patient's condition becomes serious, because something drastic has happened, or because there has been a decrease in blood pressure. The idea is to use fluids prophylactically—as a means of preventing things from happening.

I venture to say that many of you work in hospitals where fluids are not given as routine during an operation. Indeed, when visiting surgeons come here from other cities, and especially from abroad, they are startled by the fact that, even for a relatively simple operation, the anesthetist inserts a needle in a vein and hangs up a bottle of fluid for intravenous administration. They say, "Why, we have operated for a good many years, and we don't give any fluid intravenously at all!"

Our reason for giving fluids intravenously is basically to try to maintain our patients in a normal biochemical and nutritional state. This is a relatively new idea, but it is an important one. The old idea, of course, was not to do anything that was not absolutely necessary until something went wrong. For example, we did not formerly give blood until enough blood was lost to produce surgical shock. Now the idea is to know enough about disturbances in the patient's body

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and the patient's requirements to anticipate and avoid trouble. Let me mention a few more specific reasons for giving fluids during an operation.

In the first place, the effects of starvation must be combated. The patient receives nothing by mouth after midnight before the day of operation. If the operation is scheduled for noon or the afternoon, the patient may go for as long as 18 hours, sometimes longer, without fluids or food. We know that nutritional requirements are important. They include not only water but glucose and other elements, although water is the most essential. If you doubt it, try going for six or eight hours without taking anything by mouth. You would probably feel a little dry or thirsty, yet patients are often permitted to go 12 or 18 hours without fluids. If to the preoperative period is added the interval that follows operation, it is apparent that a good many hours may elapse during which the patient receives no water at all. It is necessary, then, to meet this need by starting fluid administration almost immediately.

A second reason for fluid administration during an operation is that it provides an opportunity for making the urine alkaline. Acidosis is not uncommon after long procedures, especially if the patient has circulatory impairment. It is much better to have the means available for combating acidosis—by the intravenous injection of sodium lactate or bicarbonate—than to wait until the acidosis becomes severe. The same is true of making the urine alkaline. In the presence of a hemolytic reaction, it is better to have the patient excreting alkaline urine than acid urine. Acid urine increases the tendency for hemoglobin to be deposited in the kidneys, and a renal shut-down in the presence of a hemolytic reaction may be prevented by rendering the urine alkaline.

A third reason for giving fluid intravenously during an operation is that use of the intravenous method permits the injection of certain food elements, notably glucose and perhaps amino acids. Glucose especially (when converted into glycogen) improves hepatic function. Many patients with gallbladder disease and many who are jaundiced have hepatic insufficiency. In many cases the anesthetic, especially ether, will increase liver damage. The use of any means for improving the ability of the liver to withstand these various deleterious effects will benefit the patient.

A fourth reason is that the intravenous administration of fluid corrects losses of blood and plasma. Here, again, it is better to prevent this loss or correct it while it occurs, than to wait until the patient shows harmful effects of the loss.

What sorts of fluids are used? In the old days the intravenous administration of fluids was simple. Only saline solution was given. About 1925, glucose was added to the solutions. Sodium lactate is a still more recent addition. The various fluids may be listed under five headings.

1. One kind of fluid that practically every patient requires, regardless of anything else, is

water. He needs water to maintain all vital processes. When he is deprived of water, his body becomes dehydrated. Water, of course, cannot be injected alone because it is hypotonic. Something must be added to the water to make it isotonic. The simplest element which can be added is salt, but this demands separate consideration. The next is glucose. Reduced to the simplest isotonic form, the water requirement of a patient during an operation may be met by administering a solution of 5 per cent glucose.

2. The second element that the patient needs is glucose itself, for reasons already mentioned. Glucose is needed to give energy and to improve hepatic function. Since glucose is also needed to maintain isotonicity, the complexity of the solution is not increased. Other nutritional substances, such as amino acids and vitamins, may also be added to the solution, but I doubt that they are essential during the period of the operation.

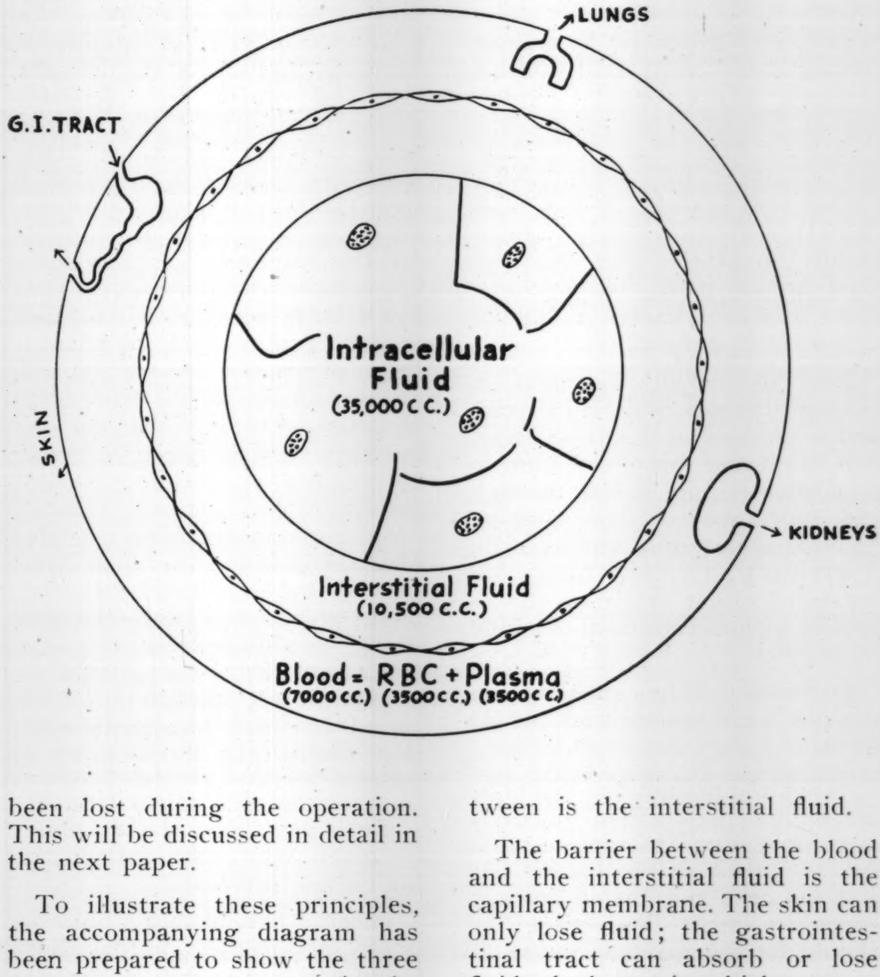
3. A third element that might be added to the solution is salt. Perhaps some of you are accustomed to giving saline solution during an operation. I question the advisability of this practice as a routine. Salt, even in a person's diet, is 90 per cent a condiment. One could reduce the salt intake to 1 Gm. a day (instead of 10) and still get along very well. Formerly salt was used to make solutions isotonic, but now glucose is used for the same purpose. Salt is necessary *only* for patients who have lost salt, primarily for those who vomit. If the patient has vomited and lost salt, he should receive—in fact, he *must* receive—sodium chloride before he goes to the operating room. Consequently, there is no need to add salt to solutions given during an operation unless somebody has failed to replace the salt lost through vomiting, diarrhea, sweating, or an intestinal fistula.

There is one occasion when salt should be added to the solution. During a long operation in hot weather, the patient might perspire so profusely that it would be wise to replace the salt lost by adding salt to the solution. That would be the only condition I know of for which salt would really be necessary during the course of an operation.

4. A fourth element might be added to the solution to combat acidosis. Sodium lactate is the one most frequently used. It has the advantage over bicarbonate in that it also provides calories and thus acts like glucose.

A solution containing all the requirements, as just outlined, is a solution of 5 or 10 per cent glucose in sixth molar lactate. This relatively simple solution contains water, glucose to build up liver glycogen and furnish calories, and lactate solution to combat acidosis. Ringer's solution, which is sometimes used, is practically the same as saline solution. It contains a little potassium and calcium, which may have some value, but it has the disadvantages of saline solution.

5. A fifth element that may be given during operations is protein in the form of plasma or whole blood. Here the problem is one of replacing an element that has



been lost during the operation. This will be discussed in detail in the next paper.

To illustrate these principles, the accompanying diagram has been prepared to show the three compartments, or spaces, in the body.

The diagram is arranged as if the human being were composed of three spheres, one inside the other (see the figure). The inner sphere is the intracellular space containing the fluid in the muscles, liver, and so forth, which is separated from the rest of the body by the cell membrane. The outer space represents the circulating blood. In the space between is the interstitial fluid.

The barrier between the blood and the interstitial fluid is the capillary membrane. The skin can only lose fluid; the gastrointestinal tract can absorb or lose fluid; the lungs, in which you are particularly interested, have no way of absorbing fluid but represent a very important way of losing fluid, e.g., water by evaporation. The kidneys also lose fluid and many other substances along with it.

The amount of fluid in each of the three spaces is quite different. Inside the cells is 35,000 cc.; outside the cells, about 17,000 cc. The latter is further sub-

divided between interstitial fluid (10,000 cc.) and whole blood (7,000 cc.). The blood, of course, contains red cells and plasma proteins. The interstitial fluid is almost simple isotonic saline solution. The intracellular fluid is protoplasm and contains about 20 per cent protein. It has been said that if more were known about the protein and the way fluids cross these two barriers, more would be known about life processes in general.

Normally, fluid is taken in through the gastrointestinal tract and is excreted in the urine, feces, and sweat and through the lungs. Abnormally, fluid is lost through the gastrointestinal tract and by hemorrhage, trauma, and so forth. Therapeutically, these losses are corrected either orally or parenterally with injections of one kind or another.

QUESTION: When would you use plasma in preference to whole blood?

When the patient has lost plasma rather than whole blood. It is always sound to replace what has been lost. If the patient has lost red cells and has an anemia, especially from hemorrage, he should be given whole blood. If he has lost plasma alone, plasma is preferable. If plasma is not available, whole blood should be given. If whole blood is not available, plasma is the best substitute. If both plasma and whole blood are available, the patient should be given what has been lost.

QUESTION: How rapidly should fluids be given?

Generally speaking, from the viewpoint of cardiovascular efficiency, the administration of fluid to an average-sized adult at a rate of more than 1,000 cc. an hour—about 20 cc. a minute—is likely to embarrass the circulation, as shown by increased venous pressure. Consequently, under ordinary conditions, fluids should be given at a rate slower than 1,000 cc. an hour. An exception to this rule is the use of fluids for the correction of circulatory impairment. If the patient has lost a large amount of blood and is in dangerous shock, blood must be given rapidly. The same is true of burn shock; plasma must often be given so rapidly that positive pressure must be used. To be accurate, one should never count the rate of fluid administration by drops per minute, unless the size of the drop is known. Some drops are twice as large as others owing to the size of the nozzle tip. The drops should first be standardized, and their size should be known before the flow is adjusted. It is the cubic centimeters that count, not the number of drops.

QUESTION: How should fluids be given when the patient has high blood pressure?

When a patient needs fluid, he needs it whether he has high blood pressure or not. In the presence of high blood pressure, fluid should be given a little more slowly. A person with a lower blood pressure could probably accommodate himself to a rapid injection of fluid, whereas a patient with high blood pressure might not adjust so well.

QUESTION: What are the dangers of administering fluid too rapidly?

Too rapid administration of

fluid will impair cardiac function; there will be too great an increase in venous pressure. Another possible danger is that the fluid may leak out rapidly through the capillaries and therefore lose its effectiveness.

QUESTION: What fluids would you give to a diabetic during a nephrectomy, and how much?

If the patient has been properly prepared before the operation, he should be treated just like an ordinary patient. He might need a little more glucose and insulin during and after the operation, but that would depend on the severity of the diabetes and of the operation. In a long procedure, the effects of the operation in general may increase the severity of the diabetic state. Trauma of any kind has often been said to produce traumatic diabetes. For this reason, one would add extra insulin or glucose during the administration of fluid.

QUESTION: Is it possible to produce shock by injecting too much fluid during the course of an operation?

I suppose it is, but I know of no data on the subject. No surgeon would admit that that would be possible. Yet I am often a little appalled to see 5 to 6 liters of whole blood, not to mention other fluids, given a patient during the course of an operation during which the loss has been only 1 to 2 liters. Whole blood contains sodium citrate which, unless rapidly metabolized, may possibly cause harm. After all, the total circulating blood does not exceed 7 liters. In the absence of actual loss, it seems to me that the administration of too much fluid might produce shock, although not in the true sense of the word. It would interfere with the cardiac and pulmonary mechanism and would thus produce pulmonary edema primarily rather than shock.

QUESTION: Why is sodium lactate preferable to sodium bicarbonate?

Sodium lactate is simply a way of giving sodium bicarbonate. Sodium lactate is metabolized to sodium bicarbonate, which then combines with the acid and therefore combats acidosis. In that change, glucose is formed. Moreover, sodium lactate is easier to prepare than sodium bicarbonate; it is stable, it can be autoclaved, and it does not have the pitfalls of preparation of sodium bicarbonate. For these reasons, the use of sodium lactate has largely displaced the use of sodium bicarbonate.

In conclusion, I am very gratified and pleased not only by the number of questions (most of which there has been too little time to answer), but particularly by the intelligent insight they show of many of the problems of fluid administration during an operation.

FLUID REPLACEMENT DURING OPERATIVE PROCEDURES

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St. Louis

I would like to preface my remarks by saying that I am just another surgeon who cares for patients. However, I belong to a unique school of thought in that I am firmly convinced that medicine is merely a means to an end. I trust that you feel that anesthesiology is a similar aid to such an objective. For the most important thing in this world is living!

Unfortunately, fluid replacement during an operative procedure is concerned with "living" —if even in a restricted sense.

I find myself in a unique position on this program, between Dr. Robert Elman, who has made most profound original contributions on the particular subject that I am to discuss today, for one phase of which he was awarded the Samuel D. Gross prize last year, and a Presbyterian minister.

In these particular days of specialization, it would seem advisable to develop some specificity of thought when the demand goes forth from the operating room, "Start some fluid!" The action follows the information, relayed by the anesthetist to the surgeon, that the blood pressure is falling, that the pulse rate is rising, that the coloring of the patient is cyanotic, or that the patient is losing more fluid by surface evaporation than is expected within normal limits, and so forth.

The competent trained surgeon knows of these changes, and he anticipates the collaboration of the competent anesthetist to keep him aware of them throughout the operations; the anesthetist's report merely confirms the opinion of the surgeon.

The request, "Start some fluid," is always given in the imperative, but there are three important points that are not usually stated, although in a few institutions where custom is followed by education, this is not necessary.

When the surgeon says, "Start some fluid," first, *what* fluid should be given?

Secondly, *where* should fluids be administered? Intravenously or subcutaneously; in the left arm or leg, or the right arm or leg?

Thirdly, and the crux, who is going to "bell the cat"! *Who*

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starts the fluid? The "circulating" nurse? An emergency man? Does someone drop out of the team, or does the anesthetist start the fluid?

I would like to develop these three very important problems with you this afternoon.

The effect of any surgical procedure may result in an abnormal, often tremendous, loss of one of several essentials—of water, of electrolytes, of red blood cells, and of plasma. The use of the anesthetic agent in certain instances may influence this acute deficit to which I have referred. In discussing the replacement of fluid, I propose to deal only with the acute deficit, that which occurs at the time of operation. It is all very simple and it is frequently correct to state that plasma and/or whole blood will promptly correct the acute deficit due to the actual loss of whole blood or plasma, to the outside, into the damaged tissues, or into the body cavities, at the time of operation. But let us go a bit further.

Two resultant conditions under consideration are sometimes difficult to separate and fall into two distinct categories, namely, hemorrage and surgical shock. It is not at all unusual to have one person say, "This is shock!" and the next person say, "Why, he is just bleeding," and, after all, the treatment is about the same, so who cares?

Coller and his co-workers[1] at the University of Michigan carefully reviewed the literature concerning the amount of blood that is lost during various surgical operations and carried on some intensive and acute experiments. They found that there is no correlation between the amount of blood loss and the changes in the hematocrit readings and the hemoglobin concentrations before and after operations. One can obtain a hematocrit reading in the operating room or serum protein determinations, but it is most difficult to judge from these two readings whether the patient will survive. Clinical judgment still takes precedence over such correlations. The lesson to be learned from Coller's experimental work and clinical observation is that a patient is benefited *most* when the blood lost is replaced with blood given *when the loss occurs.* This is my point: "Patients benefit most when the blood lost is replaced by blood given when the loss occurs," as taken from the work of Coller and others.

Now I would like to invite your attention to the table, which is reproduced from Coller's article in the *Journal of the American Medical Association.* Note, if you will, the average amount of blood loss in the usual operations. During a radical mammectomy with axillary dissection, 732 cc. blood is the loss on the average—not for the unusual, nor for the obese individual with a short, stocky neck and large breast, but for the average individual! The incidence of transfusions is, as most of you well know, rather high for the "closed" operation of prostatic revision. Such patients lose a half pint of blood on an average.

1. Coller, Frederick; Crook C. E., and Iob, V.: Blood loss in surgical operations. J.A.M.A. **126**: 1-5, Sept. 2, 1944.

Blood Loss in Operations of Various Kinds in 636 Cases Compiled from the Literature*

Operations	Number of Cases	Blood Loss Maximum, Cc.	Blood Loss Minimum, Cc.	Blood Loss Average, Cc.
Brain operations	30	2,150	487	1,084
Postganglionic neurotomy Cr.N.V.	4	650	86	337
Spinal cord operations	7	1,264	107	626
Thyroidectomies	29	1,118	16	237
Other neck operations	3	410	105	230
Mastectomies, radical	20	1,272	254	732
Mastectomies, simple	5	220	180	200
Thoracic operations	113	2,895	35	575
Biliary operations	16	400	51	100
Gastric operations	41	650	45	233
Splenectomies	2	990	160	525
Intestinal operations above sigmoid	11	230	10	81
Appendectomies	14	62	4	13
Sigmoidal, rectal and anal operations	21	1,220	8	377
Hernia operations	13	306	11	74
Miscellaneous abdominal operations	6	546	14	218
Pelvic operations	30	680	22	266
Prostatic resection, transurethral	220	1,258	4	280
Kidney operations	10	1,144	130	372
Orthopedic operations	31	1,564	40	441
Total	636			

*From Coller1

Nadal,[2] whose work was also done at Ann Arbor under Coller, found that patients who lose over 20 per cent of the blood volume show signs of clinical shock. The size of the patient is an important factor. Most patients with pyloric stenosis weigh less than 7 pounds. Fortunately, few of them bleed much, but it is apparent that a loss of 100 cc. for a baby would amount to a fifth of the total blood volume. It is possible to conclude that for all practical purposes roughly one thirteenth of the body weight is made up of blood and its elements with cells making up about 45 per cent of the volume in men and 40 per cent in women.

One must again stress the importance of admitting the fact that blood will be lost when an operation is performed; its approximate amount should be known, and the loss should be replaced with blood during the period while the blood is being lost. Consequently, there is never any excuse for calling the blood bank midway in an operation for "a unit of blood." The problem is that elementary. Because of the lack of correlation between the amount of blood lost and the simultaneous changes in hematocrit readings which result, such determinations cannot be used to estimate the need for blood volume replacement during and after an operation. One must rely primarily on knowledge of the average losses to provide a basis for the replacement of blood during an operation.

2. Nadal, J. W.: Blood loss in orthopedic operations. Univ. Hosp. Bull., Ann Arbor 5: 74-75, Oct., 1939.

Additional blood may be given if the clinical state of the patient demands it, but it is a known fact that even minimal blood loss retards convalescence, and if the surgeon and hospital superintendent are anxious to get the patient out of the hospital in the shortest possible time, the best thing to do is to replace the blood while it is being lost.

Churchill,[3] of Harvard, pointed out that the loss of whole blood from battle wounds far surpassed that estimated at the beginning of the war and that the degree of shock parallels the amount of blood loss. Many of you served under Dr. Churchill, who was surgical consultant for the M.T.O.

The replenishment of red blood cells maintains and stabilizes the required oxygen-carrying capacity of the blood. The red blood cells form an appreciable portion of the mass of blood, a portion that cannot be lost into an area within the body or into the tissues without actual disruption of the blood vessel walls. If the patient is anemic from hemorrhage, the administration of whole blood is preferable and is indicated. In the absence of hemorrhage, shock may result from the loss of the protein-containing fluid within the tissues themselves, and plasma transfusion is preferable to whole blood transfusion because plasma contains roughly twice as much protein as whole blood per unit volume. Dr. Elman has referred to this phase of shock (see page 132). Recall that red blood cells occupy space but exert no colloidal osmotic effect and cannot correct such a specific deficit as results when shock occurs in the absence of hemorrhage.

For massive hemorrhage, replacement is obviously indicated and should be carried out without delay. The acutely depleted blood volume, the lowered cardiac output, and the tissue anoxia all urgently require relief. For acute hemorrhage, plasma or a substitute injected intravenously will increase blood volume but will create further dilution of the hemoglobin and in time will cripple the mechanism for oxygen carriage. Under such circumstances, the systolic blood pressure may temporarily rise and produce a false sense of security while the patient's condition actually grows worse. When Adriani[4] presented a paper on anesthetic accidents before the members of the Southern Medical Association this year, he neglected to develop a discussion of this particular accident, hemorrhage, probably the most common anesthetic accident that occurs.

Plasma should be used to sustain the patient until blood is available. It is impossible to carry out modern surgical procedures and support the patient during the acute phase of the operation without the use of whole blood. Low titer type O blood should be used in an emergency, and the specific type and the massed blood later used when time allows. During this era of blood banks, availability of the blood is merely another detail.

3. Churchill, Edward: An American surgeon. Surg., Gynec. & Obst. **84**:530, April 15, 1947.

4. Sappenfield, R. S., and Adriani, John: Anesthetic accidents. South. M. J. **40**:455-461, June, 1947.

Venipuncture has been successfully carried out by graduate nurses for many, many years. The technic is taught in the training program for both undergraduate and graduate nurses. The particular operative set-up will vary according to the hospital, but the size of the needle employed is to some extent standardized and should be large. In each instance, as large a needle should be used as is compatible with the size of the vein in order that a free flow of blood or blood substitute may be provided if called for. The rate of flow should be steady and should be calculated to meet the current and the acute need of the particular situation.

The *time* to start fluid administration is *after* the induction of the anesthesia and just prior to the draping of the patient. Any such procedure must be started before the condition of the patient demands it and obviously prior to the beginning of the operation.

During recent years, there has been a tendency to administer subcutaneous fluids into the medial and the latter aspect of the thigh. These areas should be used only as a "last resort." The axilla is the site of choice for the subcutaneous administration of fluid when it does not interfere with the surgical procedure. The lacy, areolar tissue which fills the axilla allows mild expansion without pain, and the blood supply of the area is excellent. It must be kept in mind that the administration of fluid is of no value to a patient until the fluid has entered the circulation. Static fluid in subcutaneous tissue does not support the circulation, and such administration should be condemned.

When a hypertonic solution is used intravenously, it must be "covered" simultaneously by an adequate amount of fluid given subcutaneously to prevent dehydration as the result of diuresis. The finest diuretic agent available is any hypertonic solution given intravenously. Such is the standard treatment for suppression of urine. Thus if one gives a hypertonic solution intravenously, the patient is merely dehydrated from diuresis.

In conclusion:

1. I would like to emphasize the importance of modern day knowledge of the average requirements for standard operative procedures and again urge that replacements be made at the time of the blood losses.

2. The nurse anesthetist seems to be the logical person to "start the fluids."

3. Plasma has its place in the treatment of surgical shock which is not due to hemorrhage.

4. Five per cent glucose in saline is a suitable "vehicle" to "start the fluids" and to which blood and/or plasma are added.

5. The time to "start the fluids" is prior to operation while the anesthesia is being induced.

6. The place to "start the fluids" is in either of the branches of the external saphenous veins as they course about the ankle,

in the median antecubital vein, or in the axilla when saline is administered subcutaneously.

While everyone is relaxing and to stimulate the asking of questions in this question period, I might anticipate at least one question by saying that we are using 5 per cent glucose in saline as the fluid of choice for the original intravenous administration. Glucose is never given subcutaneously. It does not seem quite logical to increase the hypertonicity of any particular fluid. It takes very little salt to keep one in balance.

QUESTION: What should the temperature of whole blood be for transfusion? I have been asked to give it as it came from the blood bank—ice cold.

I think the ideal temperature is body temperature. That you have been asked to give blood ice cold does not affect the fact that any fluid for parenteral administration should be given at body temperature.

QUESTION: When should arterial transfusion be used?

Arterial transfusion should be used when venous transfusion is impracticable. On one occasion, I can recall giving a patient a transfusion through the aorta, the thoracic aorta. Dr. Graham had resected a rather large aneurysm in the pelvis of a woman, and there was an inadvertent loss of blood that was considerable which suddenly flooded her abdomen. Although the blood was running in from at least two points, the abdominal blood was quickly strained through gauze, and an "autotransfusion" was performed through her aorta.

QUESTION: What amount of fluid do you recommend should be given intravenously twenty-four hours postoperatively?

As our late President would say, this is an "iffy" question, and I frankly do not think I should take time to go into it because there are so many factors that enter into this problem. Let me merely say this: The important point is how much the patient voids twenty-four hours postoperatively, not the intake. The output is the important thing, not the intake. If the output is adequate, intake is assured, and at a body temperature of 39 C an output of 1,200 cc. is adequate.

QUESTION: Do you advocate periodic administration of vitamin K several days prior to surgery to prevent excessive bleeding or general oozing?

This particular drug is used for patients who are jaundiced and for those who have bleeding tendencies.

QUESTION: Of what value do you think an intravenous injection of glucose plus alcohol and B complex is to the alcoholic patient before an anesthetic is given?

I have had no experience in this particular matter. I cannot express an opinion on that question.

QUESTION: In a large hospital in the East, the surgeons allow a transfusion or intravenous injection to be given in the foot or leg only in case of an extreme emergency because of the danger of phlebitis. How do you feel?

(*Continued on page* 148)

THE INTERNIST LOOKS AT ANESTHESIA

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Prior to World War I, internists were often directly concerned with anesthesia. Not infrequently they were called upon to administer anesthetics to their own patients referred to surgeons for operation. Fortunately, for the patient, this situation no longer exists. Not only do present day internists refrain from administering anesthetics (except in relatively small communities), but the truth of the matter is that they rarely think about them. The whole problem of anesthesia is left to the surgeon and the highly trained professional anesthetist. In this connection it is interesting to note that not a single reference to surgical anesthesia appears in the index of either of the two leading American textbooks of medicine nor even in the index of the comprehensive *System of Medicine* published by the Oxford University Press. Thus, at the outset, I am inclined to question the appropriateness of the title of this article, for I am afraid the modern internist rarely gives anesthesia more than a fleeting glance.

There are, however, even in modern medical practice certain special situations in which the internist should concern himself with anesthesia. His prime concern is the toxicology of anesthetics. He must take cognizance of the potential dangers of specific drugs to patients with organic disease, for he is not infrequently called upon to assist the surgeon and the anesthetist in selecting the safest method of anesthesia in cases in which toxic reactions are likely to be serious. His opinion can be sound only if based upon a thorough preoperative examination and a knowledge of the toxicology of anesthetics.

In the selection of safe anesthesia for patients with organic disease, possible effects upon the heart, lungs, liver, and kidneys must be examined with particular care. The toxic effects of the more commonly used anesthetics upon these "vital organs" are listed in the following table. Only brief comment need be made concerning each drug.

The use of chloroform as an anesthetic has been largely discontinued because its toxic effect upon both the heart and the liver is often serious. Chloroform not only depresses the myocardium directly but not infrequently causes fatal ventricular fibrillation. Unlike most other anesthetics, chloroform, in concentra-

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TOXICOLOGY OF ANESTHETICS

ANESTHETICS	HEART	LUNGS	LIVER	KIDNEYS
Chloroform	Lowers blood pressure Depresses myocardium Lowers output Ventricular fibrillation	Slight irritation	Marked depression of function Direct liver damage	Depresses function Casts and albumin
Ether	May affect blood pressure Insignificant arrhythmias	Irritates respiratory tract Increases bronchial secretions	Slight depression of function	Depresses function significantly in advanced nephritis Casts and albumin
Divinyl ether	Unaffected	Unaffected	Depresses function Occasionally causes necrosis	Unaffected
Nitrous oxide	Anoxia* Fall in blood pressure Depresses myocardium	Unaffected except by prolonged anoxia	No significant effect	No significant effect
Ethylene	Unaffected	Unaffected	No significant effect	Unaffected
Cyclopropane	No anoxia Arrhythmias (80%) Ventricular tachycardia Ventricular fibrillation	Atelectasis	Unaffected	Unaffected
Avertin	Transient fall of blood pressure	Unaffected (Respiratory depression)	Depresses function	Depresses function Albumin and casts (Renal excretion)
Pentothal	Lowers blood pressure	Unaffected (Respiratory depression)	Detoxified by liver	Unaffected
Procaine (Spinal, caudal, local, and regional block)	Fall in blood pressure (Spinal and caudal)	Unaffected	Unaffected	Unaffected

*Prolonged use may cause permanent cerebral damage.

tions that have little or no influence upon the respiratory center, affects the heart. Chloroform also exerts a direct toxic effect upon the liver. Hepatic function is markedly depressed whenever the drug is given as a general anesthetic, and permanent liver damage may result. Kidney function is similarly though less severely affected. Chloroform is potentially so toxic that it should no longer be used for anesthesia.

Ethyl ether, on the other hand, is much safer and is still probably the most widely used of all general anesthetics. Its toxicity to the cardiovascular system is rarely serious, and it causes only slight transient depression of hepatic and renal functions. The most serious toxic effects of ether are upon the respiratory tract; the drug causes considerable irritation and in many cases brings about a significant increase in bronchial secretions. These effects are undesirable in patients with acute and chronic respiratory tract disease and account for many cases of postoperative pneumonia.

Divinyl ether appears to be even more innocuous than ethyl ether and is relatively nonirritating to the lungs. It may, however, depress liver function and occasionally causes hepatic necrosis.

Nitrous oxide is a dangerous anesthetic for patients with cardiovascular disease. Since a very high concentration of the gas itself must be used to produce anesthesia, anoxia frequently results. Prolonged lack of oxygen in the blood causes a fall in blood pressure, depresses myocardial function, and not infrequently leads to coronary insufficiency. Ethylene, on the other hand, exerts no significant toxic effects upon the cardiovascular system and may be used safely for patients with heart disease.

Cyclopropane, one of the most popular of the more potent anesthetics, causes the internist particular concern because of its action upon the cardiovascular system. Although it is needed in relatively low concentrations to produce deep anesthesia and, therefore, does not cause anoxia, it is potentially dangerous because of its tendency to produce cardiac arrhythmias. It is stated that approximately 80 per cent of patients receiving cyclopropane as a general anesthetic exhibit some form of cardiac arrhythmia. Fortunately, most of these are transient and harmless, but occasionally ventricular tachycardia develops and even fatal ventricular fibrillation. The atelectasis that occasionally follows the use of cyclopropane is thought to be due to the rapid absorption of the oxygen-cyclopropane mixture from the alveoli in areas of partial bronchial obstruction from mucus or organic disease.

Of the nonvolatile anesthetics, cocaine derivatives used for spinal, caudal, local, and regional block anesthesia are the least toxic. Except for the hypotension which may result from spinal and caudal anesthesia, no significant toxic reactions are caused by these drugs except in the very occasional patient who exhibits a true idiosyncrasy. On the other hand, avertin used as a basal anesthetic carries with it definite potential dangers. First, it not uncommon-

ly causes a decrease in blood pressure. Secondly, it depresses liver function and is thus contraindicated in hepatic disease. Thirdly, it exerts an irritating effect upon the kidneys resulting in albuminuria and depression of renal function.

The short acting barbiturates, such as pentothal sodium, are safe for most brief surgical operations except for patients with liver disease. Since these drugs are normally inactivated by the liver, they may exert a prolonged anesthetic effect in patients with poor liver function. If given too rapidly, they occasionally cause a harmful depression of the blood pressure.

This cursory review of the toxicology of anesthetics may serve as a background for a brief discussion of the selection of anesthesia for patients with complicating organic disease.

The internist's advice regarding anesthesia is most frequently sought in cases of heart disease. Patients with hypertension, recent cardiac decompensation, and coronary artery disease are known to the surgeon and to the experienced anesthetist alike to be potentially poor operative risks. Such patients occasionally die during, or shortly after, major operations. Cardiac disease, however, is rarely an absolute contraindication for surgical treatment. Even prolonged surgical procedures may be carried out if the patient is carefully prepared preoperatively, if the proper anesthetic is selected, and if the anesthetic is skilfully administered.

A number of years ago thyroidectomy was advocated for the treatment of intractable heart disease. Patients with chronic congestive heart failure and severe coronary insufficiency were subjected to thyroidectomy without immediate ills effects. Although removal of the thyroid gland proved ultimately to be of little therapeutic value, the experience gained by operating on such patients clearly demonstrated that even patients with the most severe cardiac disease can withstand both a general anesthetic and a major operation.

Of the commonly employed inhalation anesthetics, the one to be most strictly avoided in the presence of cardiac disease is nitrous oxide. The anoxia which commonly results from the administration of this gas may be exceedingly harmful and may even lead to the death of the patient. Cyclopropane, likewise, is to be avoided when possible because of its tendency to cause disturbances in cardiac rhythm. Ether and ethylene, on the other hand, may be used with comparative safety. Sudden changes in blood pressure lead to serious results in patients with hypertension or severe coronary sclerosis. Thus avertin and spinal anesthesia must be used with the greatest caution. Of particular importance in cardiac patients is the use of proper preanesthetic medication. Excitement during induction may be accompanied by dangerously sudden hypertension and can be eliminated in most cases by the judicious preoperative use of morphine or barbiturates.

The internist is also frequently

called upon to pass judgment on the use of anesthetics for patients with pulmonary disease. Here the situation is much simpler, for the only anesthetic that exerts serious effects upon the lungs is ether. Because of its irritating effect upon the respiratory tract, ether is often contraindicated. The increased moisture in the alveoli and bronchi that results from its use provides a suitable medium for the spread and growth of bacteria and interferes with the normally efficient defense of the lung against bacterial invasion. Conclusive proof of the detrimental effect of ether in pulmonary infections is to be found in the well known fact that ether anesthesia greatly facilitates the production of experimental bacterial pneumonia in a variety of laboratory animals.

Perhaps the most frequently overlooked contraindication for the use of certain anesthetics is the presence of liver disease. Chloroform, of course, should never be used, but many physicians are unaware of the dangers involved in using both ethyl and divinyl ether. These drugs depress hepatic functions, and their use for patients with infectious hepatitis or other serious hepatic disease may result in irreparable damage to the liver. Avertin, likewise, is contraindicated, and pentothal must be used only with the greatest caution.

Finally, the state of the kidneys must be considered in the selection of safe anesthesia. Avertin, for example, is not only eliminated in the urine but may depress renal function. Ether also depresses renal function, and its use should be avoided for patients with advanced renal insufficiency.

From this brief discussion it should be apparent that the internist, in spite of his ignorance of the subject in general, must occasionally concern himself with problems of anesthesia if he is to contribute to the optimal care of patients who require surgical operations. The ultimate decision as to the choice of anesthesia must rest with the surgeon and the anesthetist, but the internist by careful preoperative examination may in certain cases be able to offer valuable advice concerning possible harmful effects of anesthesia upon complicating organic disease.

FLUID REPLACEMENT

(*Continued from page* 143)

My wife gave me this necktie for Christmas. I have no choice. I think in that particular hospital they will continue to give transfusions in the extremity only in an emergency as long as the decision rests with the present chief of the surgical service. You have no choice, but many, including the surgeons in the Department of Surgery at Washington University, believe the foot is the site of choice.

In the absence of Dr. Evarts Graham, our professor of surgery, I would like to offer you a belated welcome. Most of you have read in today's newspaper that he is in London to receive the Lister Medal and to give the Listerian Oration.

NOTES

When anesthetists get together, they talk about anesthesia. They talk about gadgets, special technics, and interesting cases. This section of NOTES was originated so that anesthetists could exchange ideas in writing as they do in conversation. Send in your contribution now. Other anesthetists will be helped by it.

BUCCAL PACKS.—An edentulous patient, with hollow cheeks, often presents a problem when a mask must be applied. One solution to this problem is the insertion of buccal packs.

Strips of gauze, 12 to 18 inches long, are moistened. After the patient is unconscious, a pharyngeal airway or an intratracheal tube is inserted, and a pack is placed on each side between the cheek and jaw. This fills out the buccal spaces so that a gas mask can be applied to the face without leakage around the mask.

To prevent the gauze from slipping down into the pharynx, a stitch may be secured in the pack, brought out of the corner of the mouth, and fastened to the cheek with a piece of tape.

FILMS FOR TEACHING AIDS.—Visual education has a definite and valuable place in the field of teaching in anesthesia. The films recommended here may be used as aids in giving students a better understanding of specific subjects on the curriculum of the schools of anesthesia. To procure a film, the instructor should write to the lending agency well in advance of the date on which the film is to be presented. The films are mailed C.O.D. and should be returned promptly by prepaid express.

Physiology of Anoxia
Linde Air Products
30 East 42nd St.
New York City

This film is animated and is an excellent teaching devise for instructors who are having difficulty in teaching the physiology of anoxia.

Signs of Inhalation Anesthesia
American Medical Association
535 North Dearborn St.
Chicago 10, Ill.

This film gives a very good picture of all stages of anesthesia, from induction to respiratory arrest.

Role of Carbon Dioxide in Convulsions during Anesthesia.
American Medical Association
535 North Dearborn St.
Chicago 10, Ill.

This film is an excellent presentation of the physiologic effects of carbon dioxide accumulation. —*Marion W. Thomas, R.N.*

Fig. 1.—Top. Fig. 2.—Middle. Fig. 3.—Bottom.

DIVIDED AIRWAY.—One of the useful devices for establishing or maintaining a free airway is the English divided airway, devised by an English anesthetist who wished to remain anonymous. According to Lundy,[1] "This apparatus may be used also as a guide for the oral introduction of a Magill intratracheal tube or other similar tube when it is difficult or impossible to visualize the larynx."

Lundy has devised a handle which may be attached to the airway in much the same manner as the handle on the laryngoscope, which increases the usefulness of the airway. After the introduction of an intratracheal tube, the airway may be left in position (fig. 1), or by removing the peg, the blades may be separated and withdrawn (fig. 2). Figure 3 shows the airway with a Magill tube in place.

1. Lundy, John S.: *Clinical Anesthesia* (Philadelphia: W. B. Saunders Co., 1942) p. 279.

LEGISLATION

Emanuel Hayt, LL.B.*

LICENSING OF TECHNICIANS.—A bill was introduced in the New York State Legislature on January 21, 1948 for the licensing of medical technologists.

Medical technology is defined in the bill as "The science, art or technique of performing microscopic examinations, chemical tests, chemical analyses, colorimetric comparison tests, the culture of pathogenic bacteria on or from the blood, blood serum, feces, urine, sputum, exudates, transudates, tissues, organ contents or any by-products thereof, from the human body, living or deceased; when the tests shall be used either directly or indirectly as an aid in diagnosis or to aid in confirming a diagnosis, or to aid in the care or treatment of disease, abnormal or injured organs of the human body."

Persons desiring to practice will be granted a license upon presenting satisfactory evidence of registration as a medical technologist, either by the American Medical Technologists or the registry of technicians of the American Society of Clinical Pathologists.

Other persons will take an examination; establish that they are citizens of good moral character and graduates of an approved high school or equivalent; that they have practiced medical technology continuously for not less than two years prior to the effective date of the act; or that they have successfully completed a course of instruction in a school of medical technology approved by the department and have had not less than one year of internship or experience as a clinical laboratory technician in a clinical laboratory; or have had five years' experience and training in a clinical laboratory as a clinical laboratory technician.

The same bill also provides for the registration of clinical laboratory technicians who may be licensed upon one year of experience and training in a clinical laboratory under supervision of a practicing medical technologist prior to the effective date of the act; or upon proof of high school graduation and having completed one year of study in a school of medical technology.

Although this bill died in committee, it indicates a trend which should be watched, especially in view of the bill that was introduced two years ago for the licensing of dental assistants who administer anesthetics. That bill, which died in committee in the New York Legislature, required a course of training or approved experience in the administering of anesthetics.

In the State of New Jersey a bill similar to the one that was introduced in New York for the licensing of medical technologists was offered. This bill has not been enacted into law.

*Counsel for A.A.N.A.

Anesthetic Explosions are Legal Hazards.—The patient had been on the operating table for forty-five minutes when the anesthetic, consisting of a mixture of oxygen and cyclopropane, exploded. He was now on the critical list, reported the local newspaper, because of internal injuries caused by the explosion.

The hospital superintendent said the anesthesia mask and tubing were broken by the explosion, but that the blast did not extend to the metal tank from which the anesthetic had been flowing. He added that the cause of the explosion was unkown; all in the room at the time of the accident were uninjured.[1]

The accident undoubtedly will result in a lawsuit; the hospital, the surgeon, and the anesthetist will probably be sued, as happened recently in another case.

A patient entered a hospital to have a wart on her nose removed and her tonsils excised. She was completely anesthetized by a nurse anesthetist; the surgeon used an electric needle to remove the wart. As he was cauterizing the wound there was an explosion, described as a "flash" and a "pop" about six inches above the face and apparently within the oral and nasal passages of the unconscious patient.

It was the surgeon's plan to anesthetize the patient initially with nitrous oxide; then to interrupt the administration of such gas, and, with the anesthetizing apparatus completely shut off, to remove the wart with the electric needle; after that, without allowing the patient to regain consciousness, to anesthetize her with ether and to remove her tonsils. After the accident, he attended to the patient's injuries but did not remove the tonsils. Neither the surgeon nor the anesthetist could account for the "flash."

It appears that the "flash" resulted from the electric needle's igniting some combustible gas, either the nitrous oxide or the ether. The evidence showed four possible causes of the "flash" and explosion: the needle may have ignited (1) ether, or (2) nitrous oxide contaminated by unclean anesthetizing apparatus, or (3) nitrous oxide contaminated through an unidentified agent at some time after it was received from the manufacturer, or (4) nitrous oxide which was already contaminated at the time of purchase.

The surgeon and anesthetist both testified that the ether was not turned on at any time during the operation. The nurse in charge of surgery asserted that it was a proper and customary procedure, in order to avoid contamination, for the anesthetist to wash the breathing tube with soap and water immediately before its use; that she saw the anesthetist wash the tube.

The tank of nitrous oxide was not produced, nor was any explanation given for the failure to do so. No attempt was made to show by chemical analysis whether the gas was pure or contaminated. A doctor who testified as an expert witness suggested that it was a "possibility" or

1. New York Times, Feb. 17, 1948.

"probability" that the contamination of the nitrous oxide was the cause of the explosion; that a "spot test" could have proved whether it was entirely pure or not.

There was no proof as to whether the gas was pure or contaminated at the time of purchase, or whether the hospital used due care, after it had received the gas from the manufacturer, to keep it pure. It was not shown whether the gas would deteriorate from age or from the type of container in which it was kept; nothing was shown as to the condition of the particular container or the circumstances which existed as to its storage or when portions of its contents were extracted for use.

It was the burden of the hospital, the surgeon, and the anesthetist to show a definite cause for the accident. If they could not explain the accident, they were required to show that the accident must have been due to some unpreventable cause. The court ordered the various defendants to prove their lack of legal responsibility for the accident.[2]

CAUSES OF ANESTHETIC EXPLOSIONS

Explosions of anesthetics generally have the gas ignited from one of three causes: (1) the discharge of static electricity, (2) the cautery, and (3) the use of improper electrical equipment. Static electricity has been the principal offender and is the most difficult to control. The danger of its accumulating in sufficient quantities to form a spark hot enough to ignite the explosive gases such as cyclopropane, ethylene, ethylchloride and ether may be eliminated to a large degree by proper humidification of the air.

The second cause is the cautery. This instrument can never be safely used upon a patient anesthetized by one of the combustible gases. The use of surgical diathermy or x-ray must be considered in the same class as the cautery. The next cause of such accidents is improper electrical equipment such as lights, switches, motors, and clocks. This cause may be eliminated through the use of special equipment. Oxygen when under pressure will ignite spontaneously on contact with oil or grease.[3]

An explosion results from the combination of (1) an inflammable gas, vapor, or other substance, (2) oxygen, either pure or in the air, or a gas or other substance that provides oxygen, such as nitrous oxide, and (3) a source of ignition. Anesthetics that are inflammable and explosive include ether, ethylchloride, vinethene, and cyclopropane. Oxygen, nitrous oxide, and air are employed as diluents in the respired atmospheres. All these gases support combustion.[4]

The only anesthetic gas explosions known to have killed patients have occurred within the closed air circuit of an anesthesia machine. Unfortunately, no progress has been made toward eliminating static sparks from

(*Continued on page* 162)

2. Diermann v. Providence Hospital, et al., (Cal.) 15 CCH Negligence Cases 582.

3. Rowell, Glenn: Hospital fire hazards. Hospitals, Aug., 1940, p. 91.

4. Rovenstein, E. A.: Avoid explosions of anesthetics. Mod. Hosp., Aug., 1939, p. 72.

THE NEWS

WE DON'T KNOW OUR OWN STRENGTH

In the April 12, 1947, issue of the *Journal of the American Medical Association*, it was stated that in 1946 3,962 nurse anesthetists were employed full time and 841 were employed part time in registered hospitals throughout the country. At that time the membership of the A.A.N.A. was 3,423. Who and where are the 1,380 nurse anesthetists who are practicing in registered hospitals but who are not Association members? Why aren't they members? Is it because they do not meet the qualifications for membership? Or is it because we have failed to interest them in the Association?

To discover who these nonmember nurse anesthetists are and their whereabouts, and to determine what part of the total number of nurse anesthetists we, as an association, actually represent, the Executive Office of the A.A.N.A. is going to conduct a survey in which each Association member is being asked to co-operate. As a start, each Association member is requested to send to the Executive Office the names, the home addresses, if known, and the hospital affiliations of all nurse anesthetists in her hospital or community who are not members of the Association.

FIFTEENTH ANNUAL MEETING

Headquarters for the Fifteenth Annual Meeting of the A.A.N.A., in Atlantic City, September 20-23, will be the Ritz-Carlton Hotel. (See page 157 of this JOURNAL for form to be used to obtain application for reservations.) The meeting of the Assembly of Schools of Anesthesia is being planned for September 20. The program for the general scientific sessions is being prepared by the Program Committee, Alma G. VanGorden, chairman, Eva McArthur, and Verna Bean. Local arrangements are being handled by Dorothy N. Ball, chairman, Marie Glick, and Olive May Murphy.

The business session is being scheduled for Tuesday, September 21, at which time there will be further discussion of the proposed revisions of the bylaws and the election of officers. At the banquet to be held on Wednesday, September 22, the second award of appreciation will be presented.

The complete program for the convention will be published in the August JOURNAL.

AMA BOARD OF TRUSTEES MOTION ON NURSE ANESTHETISTS

"The attention of the Board was called to articles being published in the lay press regarding nurse anesthetists, and it was voted to condemn publicity that is not based on a scientific understanding and that does not accurately reflect the prevailing situation."—from a Condensed Report of the Meeting of the Board of Trustees, J.A.M.A., **136:** 834, March 20, 1948.

TEXAS GIFT TO CHINA

At the twelfth annual meeting of the Texas Association of Nurse Anesthetists in Dallas, March 4-6, a contribution of $100 was voted to the American Bureau for Medical Aid to China. Dr. Minnie Maffett, professor of clinical gynecology at Southwestern Medical College and a member of the Bureau, said that "In the field of human needs there is no race, color, or creed. No one knows better than you that disease knows no nationality, no territorial boundaries—that neither oceans nor mountains are barriers. For health there is but one world. To meet some of these needs in that war torn country China, the American Bureau of Medical Aid to China was founded in 1937 by two Chinese physicians and one Chinese business man. They were soon joined by American leaders in medicine and public health, for the purpose of bringing medical aid to China, and to help China develop her own medical services . . . . In helping China, helping her to a higher level of health, we are again reaffirming that we are our brother's keeper, and that we have an unshaken faith that, in health and human welfare, *this* is One World."

Re-elected to office at this meeting were Jessie L. Compton, president; Laura Hoffman, vice president; Mrs. Jack K. Childress, secretary-treasurer; Virginia Futch, trustee.

GIFT TO SCHOLARSHIP FUND FROM TENNESSEE ASSOCIATION

At the Mid-South Assembly of Nurse Anesthetists, $200 was donated to the scholarship fund of the American Association of Nurse Anesthetists by the Tennessee Association of Nurse Anesthetists, at a meeting held at the Hotel Peabody on February 11. Gertrude A. Troster, a member of the Tennessee Association, is chairman of the A.A.N.A. Scholarship Fund Committee.

Theresa W. Trail, President
Tennessee Association of Nurse Anesthetists

CALENDAR OF COMING EVENTS

May 3-5	Tri-State Assembly of Nurse Anesthetists, Chicago
May 11-12	Fourteenth Annual Meeting, New York State Association of Nurse Anesthetists, Hotel New Yorker, New York City
May 15	Annual Meeting, Michigan State Association of Nurse Anesthetists, Mt. Carmel Mercy Hospital Detroit
May 31—June 4	Biennial Convention of the ANA,NLNE, and NOPHN, Chicago
June 2-4	Upper Midwest Allied Hospital Conference and Assembly of Nurse Anesthetists, City Auditorium, Minneapolis
September 20-23	ANNUAL MEETING, AMERICAN ASSOCIATION OF NURSE ANESTHETISTS, Ritz-Carlton Hotel, Atlantic City

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At this meeting the following officers were elected: Theresa W. Trail, president; Nancy Kittle, 1st vice president; Gertrude O'Kelley, 2nd vice president; Laura L. Grube, secretary; Shirley Price Lehman, treasurer; Alice Sims, historian; Ruthie Hawne, Hattie Vickers, Ora L. Clarke,. and Helen Whaley, trustees.

ACQUISITIONS TO LIBRARY

The Library of the American Association of Nurse Anesthetists is open to members for reference work. Recent acquisitions include the following books received as gifts and for review.

Adams, R. Charles: ***Intravenous Anesthesia*** (New York and London: Paul B. Hoeber, Inc.) 1944.

Adriani, John: ***The Chemistry of Anesthesia*** (Springfield, Ill.: Charles C Thomas, Publisher) 1946.

Adriani, John: ***Techniques and Procedures of Anesthesia*** (Springfield, Ill.: Charles C Thomas, Publisher) 1947.

Clement, F. W.: ***Nitrous Oxide-Oxygen Anesthesia*** (Philadelphia: Lea & Febiger) 1945.

Crile, George: ***George Crile: An Autobiography*** (Philadelphia and New York: J. B. Lippincott Co.) 1947.

Guedel, Arthur E.: ***Inhalation Anesthesia*** (New York: The Macmillan Co.) 1937.

Hayt, Emanuel, and Hayt, Lillian R.: ***Law of Hospital, Physician and Patient*** (New York: Hospital Textbook Co.) 1947.

Karch, R., and Estabrooke, Edward C.: ***250 Teaching Techniques*** (Milwaukee: The Bruce Publishing Co.) 1943.

Larsell, Olof: ***Anatomy of the Nervous System*** (New York and London: D. Appleton-Century Co.) 1942.

Macintosh, R. R., and Bannister, Freda B.: ***Essentials of General Anaesthesia*** (Springfield, Ill.: Charles C Thomas, Publisher) 1945.

Macintosh, R. R., and Mushin, William W.: ***Local Anaesthesia: Brachial Plexus*** (Springfield, Ill.: Charles C Thomas, Publisher).

Nash, Joseph: ***Surgical Physiology*** (Springfield, Ill.: Charles C Thomas, Publisher) 1947.

Raper, Howard Riley: ***Man against Pain*** (New York: Prentice-Hall, Inc.) 1945.

Read, Grantley Dick: ***Childbirth without Fear*** (New York and London: Harper & Bros., Publishers) 1944.

Robinson, Victor: ***Victory over Pain*** (New York: Henry Schuman) 1946.

Rowbotham, Stanley: ***Anaesthesia in Operations for Goitre*** (Springfield, Ill.: Charles C Thomas, Publisher) 1946.

Seldin, Harry M.: ***Practical Anesthesia for Dental and Oral Surgery, Local and General*** (Philadelphia: Lea & Febiger) 1947.

White, James C., and Smithwich, Reginald H.: ***The Autonomic Nervous System*** (New York: The Macmillan Co.) 1947.

UPPER MIDWEST CONFERENCE

The Upper Midwest Hospital Conference, composed of hospital associations and allied groups from Minnesota, Iowa, North Dakota, South Dakota, Montana, and the province of Manitoba, will organize in Minneapolis, June 2-4. The annual meeting of the Minnesota Association of Nurse Anesthetists will be held in conjunction with the conference at the Minneapolis Auditorium. Visiting anesthetists are invited to observe the anesthesia departments in Minneapolis and St. Paul Hospitals on Wednesday, Thursday, and Friday morning. The Minnesota Association is planning an educational exhibit, to which members are asked to contribute materials and ideas; Martha Lundgaard, Minneapolis General Hospital, is arranging the display.

EIGHT QUALIFYING EXAMINATION

The eighth qualifying examination will be held on November 15, 1948. Applications *must* be received not later than October 1. The application fee is $3.

EMERGENCY OVER

State associations which have made exceptions in favor of service personnel in the payment of dues are notified that the national emergency will be considered officially over on May 31, 1948.

MAKE YOUR HOTEL RESERVATIONS NOW!
for the
FIFTEENTH ANNUAL CONVENTION
September 20-23, 1948
Atlantic City

Applications for hotel reservations may be obtained by filling out this form and returning it to:
The Executive Office
American Association of Nurse Anesthetists
22 East Division St.
Chicago 10, Ill.

Name..

Address...
(street) (city) (state)

If you wish to be assigned a room with another person, please give her name below:

Name..

Address...

She is.............., is not.............., a member of the Association.

She has.............., has not.............., expressed her willingness to share the room.

EACH PERSON MUST FILE A SEPARATE APPLICATION.
APPLICATIONS SHOULD BE MADE BEFORE JULY 1.

ABSTRACTS

LIVINGSTONE, H.; LIGHT, G.; COTO, J., AND ENGEL, R.: Anesthetic mortality in intrathoracic surgery. Arch. Surg. **55**:545-556, Nov., 1947.

"Intrathoracic surgery is accompanied with many unusual surgical and anesthetic hazards. . . . Because of the nature of the lesions, a large percentage of the patients are dehydrated and undernourished, anemic, coughing and dyspneic and have other manifestations of serious alterations in their cardiorespiratory mechanisms. Because of the serious anesthetic and surgical risk, it seemed timely to investigate the outcome in intrathoracic operations and to determine the relationship of the deaths to the anesthetic management. The series of 688 intrathoracic operations presented here represents the work of nine surgeons and twenty physician-anesthetists.... The greatest number of deaths (15) occurred one to three days postoperatively . . . . There were no deaths due to the anesthesia alone. Of the deaths, 11.86 per cent, or 1.01 per cent in the total series, were due to a combination of anesthesia and operation, while 15.26 per cent (or 1.3 per cent in the total series of operations) were due to the operation per se. Twenty-three and seven-tenths per cent (2.03 per cent of the series) were due to the operation and the disease, and the same percentage were due to the disease alone. Of the 14 deaths grouped under 'sepsis or late surgical accidents,' 5 were due to sepsis, 1 to morphine overdosage, 1 to uremia from sulfathiazole, 2 to uremia from nonanesthetic causes, 4 to separation of sutures and 1 to suicide, making up 23.73 per cent of the total deaths or 2.03 per cent in the series of 688 operations." [In the series of 688 intrathoracic operations, there were 59 deaths, or 8.57 per cent.]

LENAHAN, NORRIS E.: Anesthesia in thoracic surgery. Arch. Surg. **56**:14-20, Jan., 1948.

"With . . . improvements in anesthesia, many surgical procedures which at one time were not possible can now be done with ease. In my opinion, three of the most important technical advances are: first, the routine use of the endotracheal tube; secondly, the use of controlled respiration; and thirdly, adequate blood or fluid replacement . . . . In discussing anesthesia for thoracic surgery I shall limit my remarks to two main types of operations: namely, lobectomy and pneumonectomy. These patients usually differ from those in the average surgical case. They usually show prolonged toxemia of amyloid disease and require an anesthetic of low toxicity and high oxygen concentration. Their vital capacity is usually seriously decreased, and there is usually much sputum and often blood in their tracheobronchial tree . . . . In the hands of my colleagues and myself induction with sodium pentothal followed by curare and cyclopro-

pane, oxygen and helium has been the anesthetic of choice . . . . The three most important principles in anesthesia in thoracic surgery . . . are: (1) Adequate oxygenation by controlled respiration. (2) Physiologic positioning which would be supine or flat on the back because of (a) least amount of mediastinal shift; (b) minimum amount of positive pressure for effective respiration; (c) minimal flooding of infected sputum to opposite lung. (3) Adequate replacement of blood volume, preferably with blood, secondly plasma, and lastly with crystalloids. Tracheal toilet is carefully performed at the end of each operation to assure an open airway and freedom from blood, mucus or plugs of various kinds."

DIPPEL, A. L.; HELMAN, R. J.; WOLTERS, C. E.; WALL, H. A., JR., AND HAIRSTON, F. H.: Sodium pentothal anesthesia for selected vaginal obstetrics. Surg., Gynec. & Obst. **85**:572-582, Nov. 1947.*

"The use of intravenous sodium pentothal in obstetrics is not new. . . . Our use of intravenous sodium pentothal anesthesia for vaginal delivery was instituted during World War II because of the shortage of anesthetists available for routine ward deliveries. . . . Between July 24, 1945, and October 1, 1946, 350 patients from both ward and private services of the Hermann Hospital were delivered of viable pregnancies under intravenous sodium pentothal anesthesia. Ward patients selected for this type of anesthesia were those in whom there was every reason to believe that delivery of the child could be completed promptly after induction of anesthesia, i.e., before the usual time required for an appreciable amount of the barbiturate to reach the fetal circulation. . . . The material used was provided in a two-ampoule carton. One ampoule contained 1 Gm. of sodium pentothal powder and the other 50 cc. of sterile distilled water from which the 2 per cent solution was freshly prepared for each patient. The special sets kept handy for this use contained two 20 cc. syringes, rubber tubing with adaptor for syringe and needles, and three needles—one no. 18 and two smaller needles. A metal syringe holder was clamped to the side of the delivery table and the patient's arm fixed to the armboard. The patient was prepared in lithotomy position and draped for delivery. The bladder was generally catheterized during the process of induction of anesthesia. The accoucher designated when he was ready to proceed with the actual delivery. An anticubital vein was usually selected for puncture and the vein catheterized in order to reduce the possibility of extravenous injection. Two or 3 cc. of the 2 per cent solution was injected initially. The patient was then asked to count out loud and an additional 2 or 3 cc. injected every 20 to 30 seconds until the patient ceased to count, after which an additional 5 cc. was given. By this time, the patient was generally ready for vaginal examination or delivery. At the slightest movement on the part of the patient, an additional 5 cc. of the drug was injected. An airway was used only when anesthesia was quite deep. If apnea

*Read before the Texas Association of Nurse Anesthetists, Houston, March 28, 1947.

appeared, the injection was stopped until respirations began again. . . . The level of anesthesia was generally not deep and respiratory efforts were moderately full. Similarly, oxygen administered until after delivery of the child was abandoned as unnecessary for such brief periods of anesthesia. . . . The umbilical cord was clamped promptly after delivery in order to make certain that the flow of pentothal from mother to child ceased. . . . we are not claiming any outstanding advantages for intravenous sodium pentothal anesthesia even in selected vaginal deliveries and are neither using nor recommending it as a routine obstetrical anesthetic. . . . As used in a small series and in 2 per cent solution, we have found it safe for mother and child. . . ."

LEGISLATION

(*Continued from page* 153)

within the machine itself. Theoretically, the safest method would be to ionize the gas within the machine's closed circuit. If this were done, the air itself would become a conductor of static electricity and continually neutralize any potential. Experiments are in progress on comparatively long-lived alpha ray emitters which eventually may lead to the development of an absolutely safe anesthesia machine.[5]

Responsibilty in Anesthesia

Routine rules against the explosion hazard from anesthetics

5. Brick, George H.: Control of anesthesia hazards. Mod. Hosp., Feb., 1948, p. 63.

should be adopted and posted in each operating room corridor of the hospital. The anesthetist and operating room supervisor are responsible for seeing that the rules are carried out. Aside from the fact that such regulations may aid to prevent accidents, they will provide some evidence of due care on the part of the hospital.

Unless it can be shown that the anesthesia apparatus was defective or that the anesthetist employed by the hospital was lacking in training and experience, the hospital would not be liable for the anesthesia death of a patient.

There is in hospitals a triple responsibility: the surgeon is chargeable with the professional care of the patient; the anesthetist, for the anesthetic she is giving under his orders; the surgical supervisor is under a duty to see that strict surgical technic is observed, that regulations are obeyed, and that the schedule of operations proceeds as smoothly as possible. In addition, there is always the possibility that the hospital itself will be held liable for injury to the patient.[6]

It is the duty of the anesthetist to superintend the preparation of the anesthesia machine and to test it before use. The surgeon is responsible for the operative procedure and its results; the anesthetist is responsible for the life of the patient so far as the anesthesia is concerned.[7]

6. Hardgrove, A. E.: Organization and administration of a department of anesthesia. Hospitals, June, 1936, p. 119.

7. Stonakar v. Big Sisters Hospital, 2 P. 2d 520 (Cal.)

BOOK REVIEWS

INHALATION ANESTHESIA, A FUNDAMENTAL GUIDE. By Arthur E. Guedel, M.D., Associate Clinical Professor of Surgery (Anesthesia), University of Southern California School of Medicine. Cloth. 172 pages, 6 charts. New York: The Macmillan Co., 1937. $4.00.

Probably no reader among the members of the A.A.N.A. needs an introduction to this book. It is a "must" on the library shelves of schools and of individuals working in the field of anesthesiology. It is presented here merely to recall to the attention of nurse anesthetists the valuable, basic information it contains on the signs and stages, mechanisms, and hazards of anesthesia.

ANAESTHESIA IN OPERATIONS FOR GOITRE. By Stanley Rowbotham, M.D., D.A., Honorary Anaesthetist to the Royal Free Hospital, to Charing Cross Hospital, and to the Royal Cancer Hospital. Cloth. 104 pages, 54 illustrations. Springfield, Ill.: Charles C Thomas, Publisher, 1946. $4.50

This monograph covers the subjects of the anatomy, pathology, examination of the patient, and diagnosis of diseases of the thyroid. Chapters on the premedication, local and general anesthesia, and the postoperative care follow in order. The text, especially the chapters on anesthesia, is liberally illustrated with pictures and drawings. The index is brief but adequate. The methods described are those used in the Thyroid Clinic of the Royal Free Hospital, London. The author does not claim that the methods which he describes are the only means of obtaining good results.

MAN AGAINST PAIN. By Howard Riley Raper. Cloth. 337 pages, 26 illustrations. New York: Prentice-Hall, Inc., 1945. $3.50.

This history of anesthesia covers the subject from historical references to man's search for relief of pain through World War II. It is written in a style attractive to the nonmedical reader and may be factually useful to those studying anesthesia. Preceding the text are a series of pictures pertinent to the subject. A list of selected items pertaining to the subject of the book is followed by a lengthy critical bibliography. In this section the author compares and comments on the pros and cons of the controversial issues which still exist in the history of anesthesia. A general bibliography and an index complete the volume.

250 TEACHING TECHNIQUES. By Lt. R. Randolph Karch, U.S.N.R., Principal, High School of Graphic Arts and Printing, Cincinnati; and Lt. (jg) Edward C. Estabrooke, U.S.N.R., Officer-in-Charge Teacher Training, Navy Pier, Chicago. Cloth. 131 pages, 4 sketches, 2 charts. Milwaukee: The Bruce Publishing Co., 1943. $1.25.

Here is a concise presentation of the solutions to problems which confront instructors. The book is small and does not elaborate at great length on any one subject. It was written during World War II as a guide to per-

sons upon whom the duty of teaching was placed, often without preliminary preparation. For this reason, it could well be used by instructors in the field of anesthesia. The 250 technics are listed in the table of contents. Each one is discussed in a short paragraph. Ten major divisions of the book give the patterns for the qualities of a good instructor, conduct of shop activities, the planning and presentation of a lesson, conduct of demonstrations, use of oral questions and discussions, motion pictures and film strips, the writing and use of tests, maintenance of good discipline, the planning of a course of study, and the writing of instruction sheets. A brief index recapitulates, in alphabetical order, the material in the table of contents. Directors of schools, instructors, and persons preparing examinations will find this book useful and informative and without the burden of great detail.

ANATOMY OF THE NERVOUS SYSTEM. By Olaf Larsell, M.A., Ph.D., Sc.D., Professor of Anatomy, University of Oregon Medical School. Cloth. 443 pages, 341 illustrations. New York: D. Appleton-Century Co., 1942. $8.00.

This book is primarily an aid to the study of the structure of the nervous system. Physiology is included when such an addition aids in an understanding of the anatomy. The material is not presented for special use in the study of anatomy as related to anesthesia, but it should be of great value as a reference for those interested in the teaching or the study of anesthesiology. The bibliography is presented in two parts, general references and footnote references, each of which follows the text. At the end of some chapters, there are listed illustrative lesions involving the portion of the nervous system which has been discussed. The index is compiled with both nouns and adjectives, which makes it useful to the novice on the subject.

CONTROL OF BREATHING

(*Continued from page* 103)

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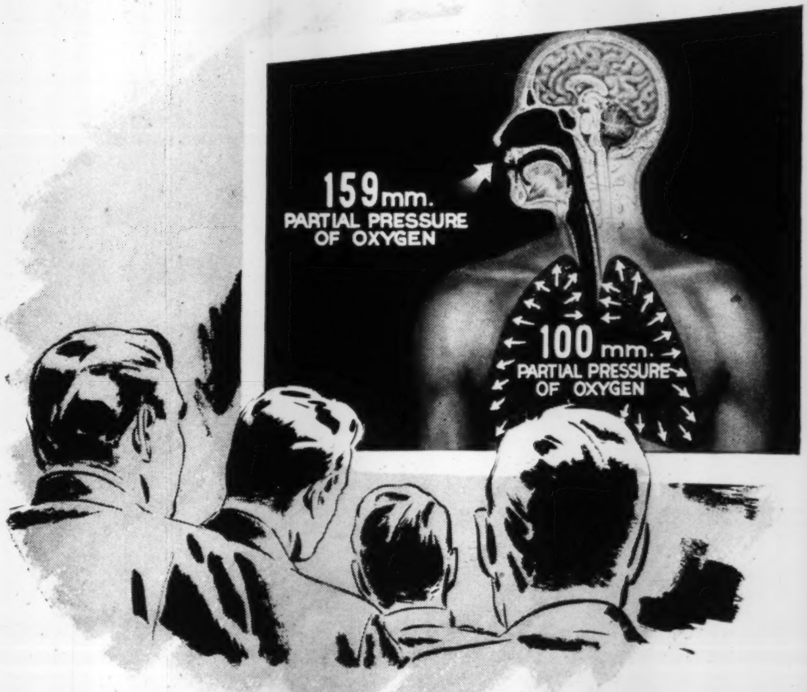
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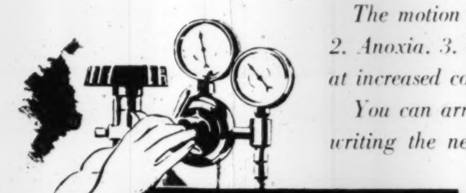
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